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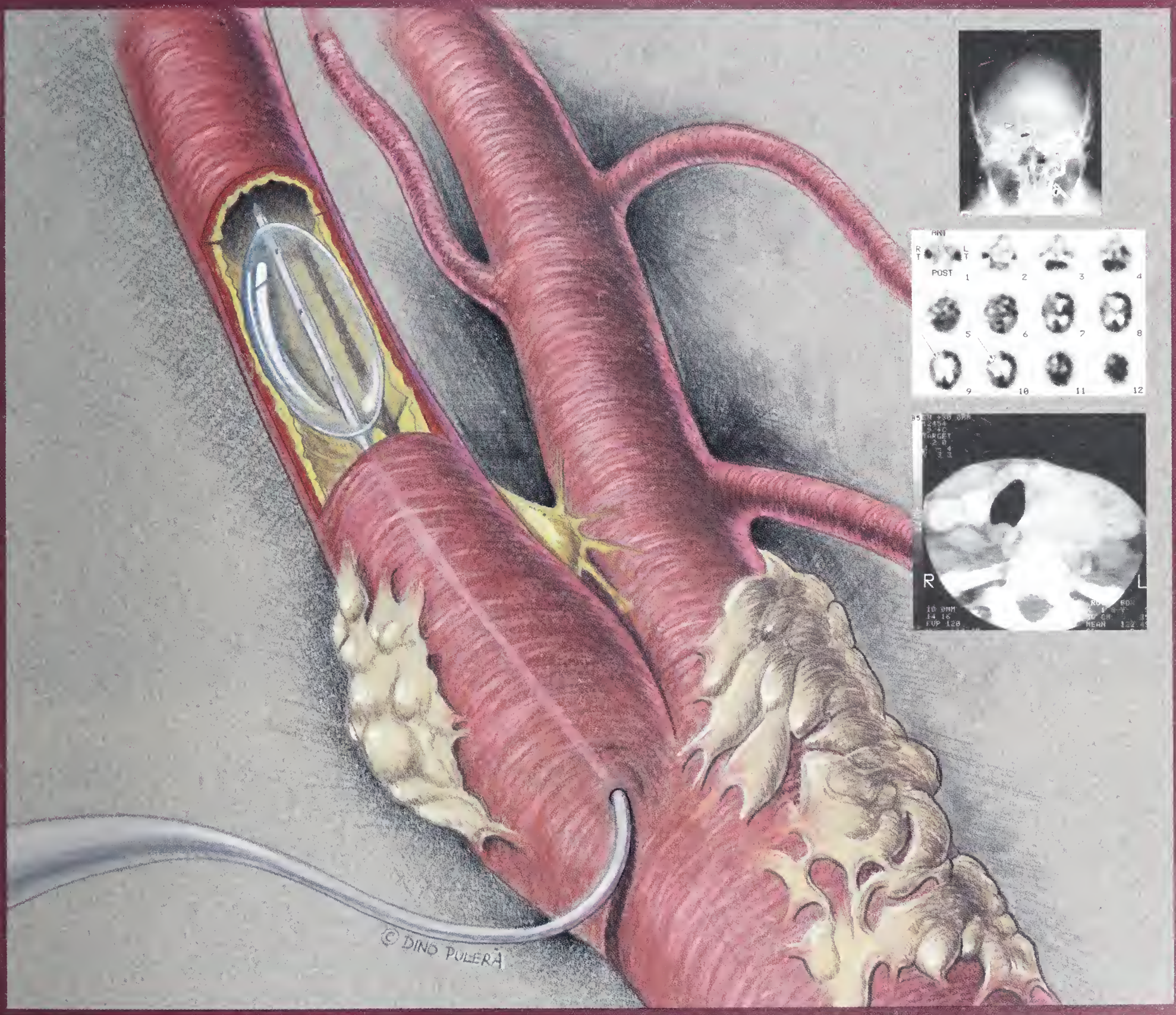

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- Test Occlusion of the Carotid Artery
- Dysphagia
- Blepharoptosis
- Pilonidal Sinus Disease

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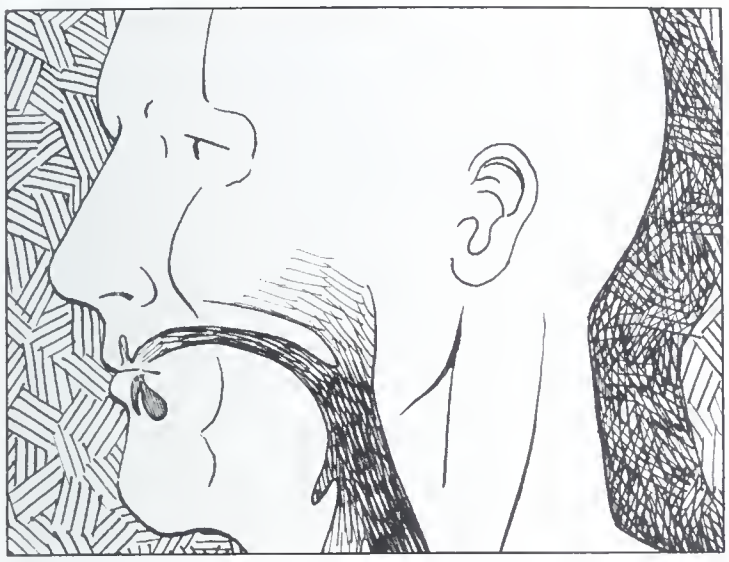

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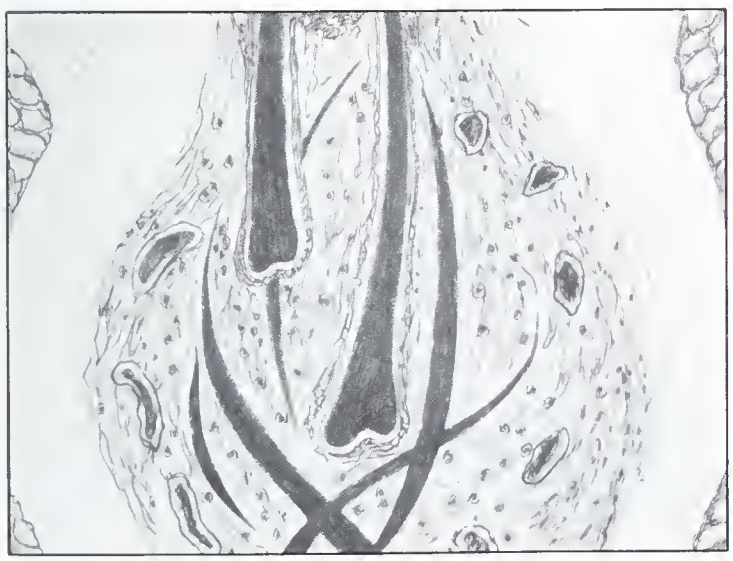

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Front cover illustration by Dino Pulera,
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Editorial

Dear Readers,

Evolution, a mechanism of continuing change, is a process we would like to reflect upon in this issue of the Journal. Seventy years ago, after the 2nd edition of the University of Toronto Medical Journal, the Editors felt it was a "fit occasion for taking stock". The Journal began as an experiment... a magazine. It was hoped that the Journal would grow both in size and in circulation. It was an ambitious goal to have the Journal become a medium for the publication of student articles; at that time less than half of the articles were by student authors.

We are proud of the evolution of this publication from an experiment to a journal in which nearly every article has student authorship; a journal containing outstanding illustrations and cover artwork; a journal which is supported by over 300 patrons, with a circulation of several thousand to countries as far away as Japan and India; a journal that has survived the test of time to become Canada's oldest undergraduate student publication.

In addition to research papers in a diversity of areas such as surgery, medicine, basic and social sciences, and ethics, the Journal also publishes articles on International Health and Community Health. As well, regularly published columns include a *Book Reviews* section, a *News and Views* section which highlights areas of medical news in Canada and around the world, a *Quick Diagnosis* section which serves as a self-test for the reader, and a *Looking Back* section in which interesting and provocative past Journal articles are reprinted. For the *Looking Back* section in this issue we have chosen an article describing the diagnosis and treatment of Tuberculosis. Such a reprint is timely considering resurgence of TB in recent years, and fits well with the theme of evolution; the reader will surely agree upon perusing the "Treatment" section of this article.

The research papers published in this issue cover an eclectic mixture of topics including dysphagia, blepharoptosis, and pilonidal sinus disease. Each of these articles provide the reader with a portrait of the condition as well as treatment and management options. Our feature article reviews some of the available tests performed to assess patient tolerability of carotid artery occlusion, an important part of minimizing complications of this procedure.

Finally, in keeping with our focus on progress, we are pleased to announce that commencing with this issue, the Quick Diagnosis section will be associated with one prize of $50.00 to the reader who provides the correct diagnoses for all cases described in a particular issue.

We hope you enjoy this first issue of the 72nd edition, and we encourage your participation in the longstanding tradition called the U of T Medical Journal.

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Test Occlusion of the Carotid Artery Before Surgical Sacrifice: A Review

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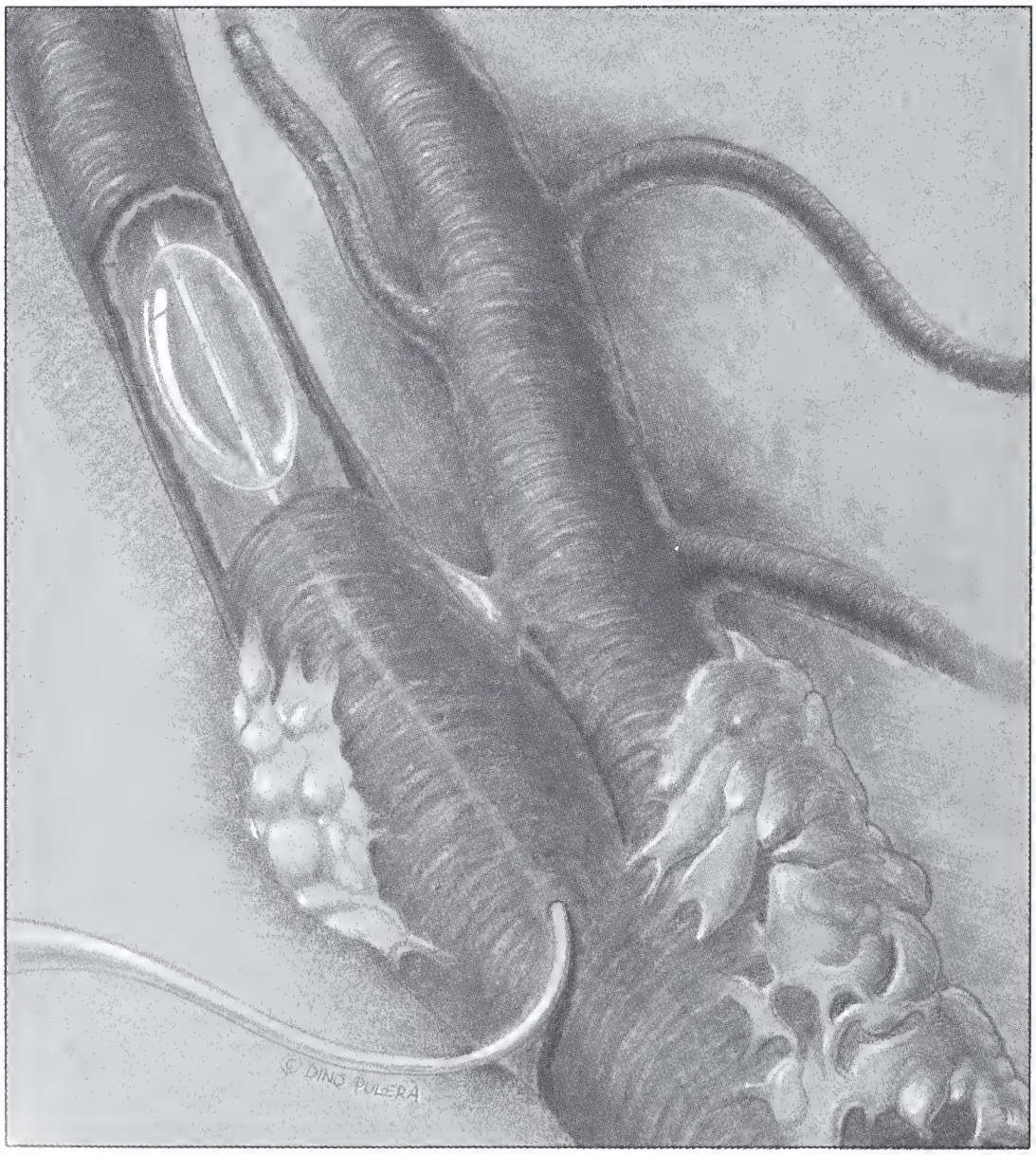
Abstract

Both benign and malignant neoplasms of the head and neck have been found on occasion to encompass or even invade the carotid artery. The morbidity and mortality associated with carotid artery ligation often prevents proper tumour removal with clear margins. Over the years, methods to assess the tolerance of carotid occlusion have been developed. These pre-operative tests often allow surgeons to identify those patients who are able to undergo carotid artery occlusion with minimal likelihood of complications. In this paper we attempt to identify and describe the more common and more sensitive tests available in assessing carotid artery ligation. In particular, at this institution we recommend the use of Xenon-CT to accurately identify those patients who will tolerate carotid artery occlusion.

Introduction

Ligation of the great vessels in the neck has historically been performed for a variety of reasons including headaches, epilepsy, haemorrhage and even hemiplegia. John Abernathy was the first person to describe carotid artery ligation. He originally ligated the carotid artery in response to haemorrhage secondary to trauma[1]. Presently, ligation of the common carotid artery is indicated for haemorrhage from the carotid artery, aneurysms or arteriovenous fistulas and for head and neck neoplasms invading the carotid artery. However, this procedure is still associated with significant morbidity and mortality, most commonly due to cerebrovascular complications.



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Many tests have been devised over the years to assess the risk of morbidity involved with carotid artery ligation. These procedures vary in invasiveness, technology and most importantly in their validity. For example, Watson and Silverstone[2] reported on the use of the Crile clamp (Figure 1) to slowly occlude the carotid artery. If any symptoms were noticed over a 24 hour period, then the clamp was either loosened or removed completely. This represented one of the first documented accounts of test occlusion and subsequent permanent carotid artery occlusion. This paper will outline some of

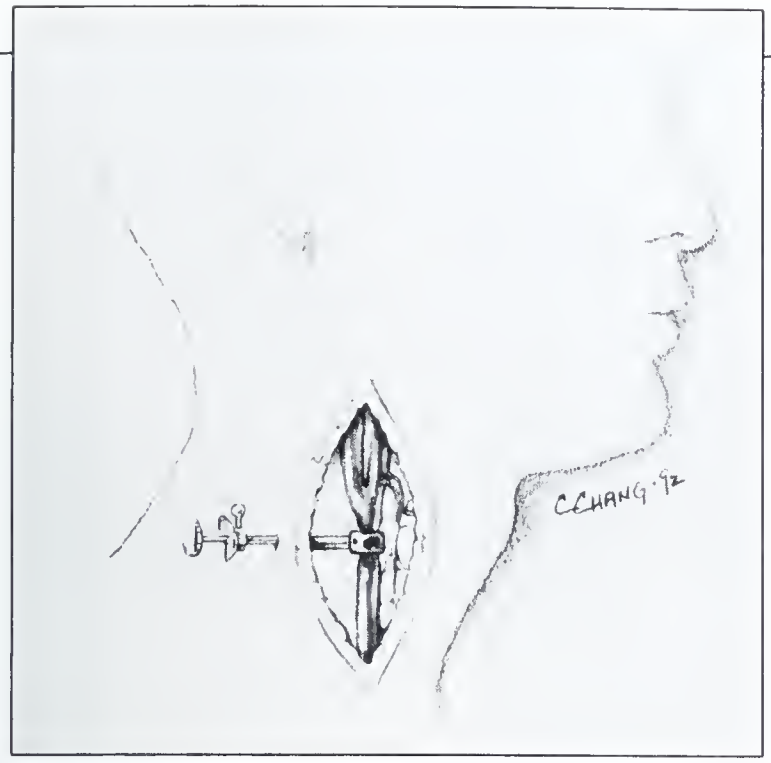


Figure 1: Insertion of Crile Clamp on right common carotid artery.

the more common and improved techniques for assessing the morbidity of carotid artery ligation.

Anatomy and Physiology

The brain, although comprising only one kilogram of the body weight, requires approximately 20% of the blood supply as well as 20% of the total body oxygen consumption. It has been found that normal cerebral blood flow (CBF) is about 55ml/min/100g of brain tissue. The critical CBF at which irreversible neurological damage occurs is approximately 20ml/min/100g. There is a three-fold amount of cerebral flow reserve before hypoxic injury occurs. This flow reserve maintains perfusion above the critical value in periods of stress such as hypotension, diuretic use or other physiologic insults causing hypovolemia. This concept of flow reserve becomes essential in evaluating patients undergoing carotid artery occlusion.

The vascular supply to the brain is delivered to the organ by two main arterial pathways. About 90% of blood originates from the paired carotids, while the remaining 10% is supplied by the vertebrobasilar system. Generally, no direct collateral flow exists between the vertebral and occipital or the carotid and vertebral systems. In one percent of patients, however, collateral flow may develop among these vessels. These two vessel systems, the carotid and vertebrobasilar, are integrated in a common anastomosis known as the Circle of Willis. The Circle of Willis (Figure 2) represents the most important collateral circulation within the brain. Here, the posterior cerebral artery (PCA) originates from the vertebrobasilar system while the internal carotid artery gives rise to both the anterior and middle cerebral artery (MCA). The communicating arteries provide collateral flow when one of the main cerebral arteries has a proximal occlusion. The circle varies widely, with only 20% of people having a "normal" Circle of Willis. With obstruction of the internal carotid artery, the flow through the contralateral internal carotid and vertebral artery is increased by 20% to compensate for the occlusion. In addition, during common carotid artery occlusion, the ipsilateral external carotid also provides minor retrograde flow to the brain[3].

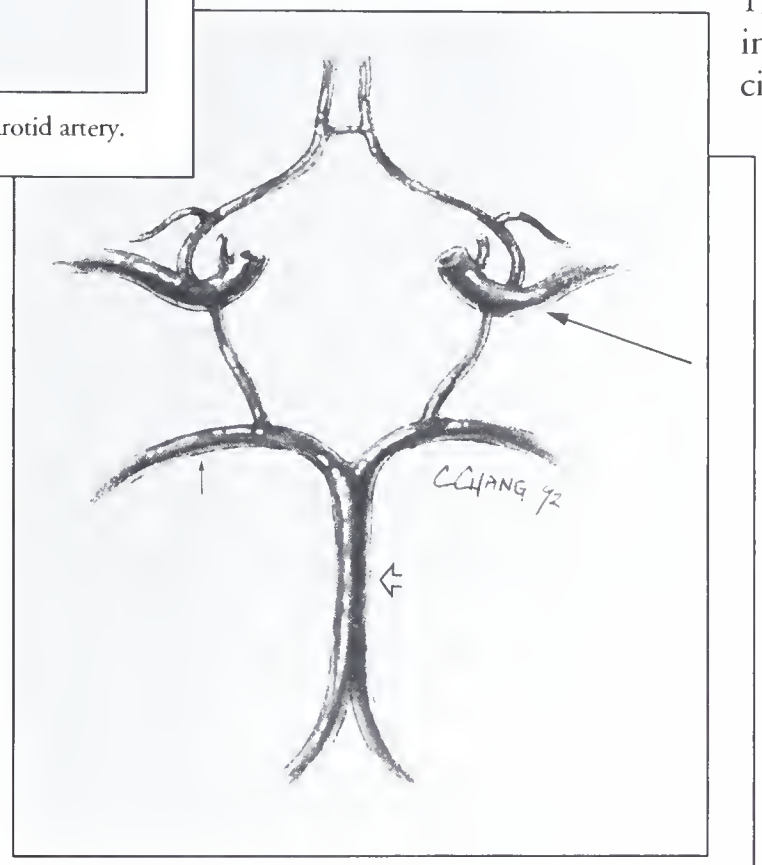


Figure 2: "Normal" Circle of Willis. Basilar a. (open arrow head), posterior cerebral a. (short arrow), internal carotid a. (long arrow).

Matas Test

Rudolph Matas was an american general surgeon who practised in the late 19th and early 20th century. Even in 1914 Matas realized the importance of collateral circulation. He related: "We have now advanced sufficiently in our methods of testing the efficiency of the collateral circulation before undertaking operations on the blood-vessels, to abandon as obsolete and pernicious the old empiric practice of trusting blindly to the admitted resources of the collateral circulation."[4]. Matas described two tests used in assessing the collateral circulation. The first was simple compression of the artery using an elastic bandage. When applied to the main artery, changes were observed which would be consistent with poor blood flow indicating inadequate collateral perfusion. This test was most commonly used on accessible arteries such as those in the extremities. The second test was reserved for either the common or internal carotid artery. Pliable aluminum bands were surgically placed around the vessels until no distal pulsatility was observed. If the patient tolerated the arterial occlusion the clamps would remain. Once signs of vascular compromise were observed, the wound would be re-opened and the clamp released. This was done within the ensuing 56 to 72 hours as no damage would be inflicted upon the arterial wall.

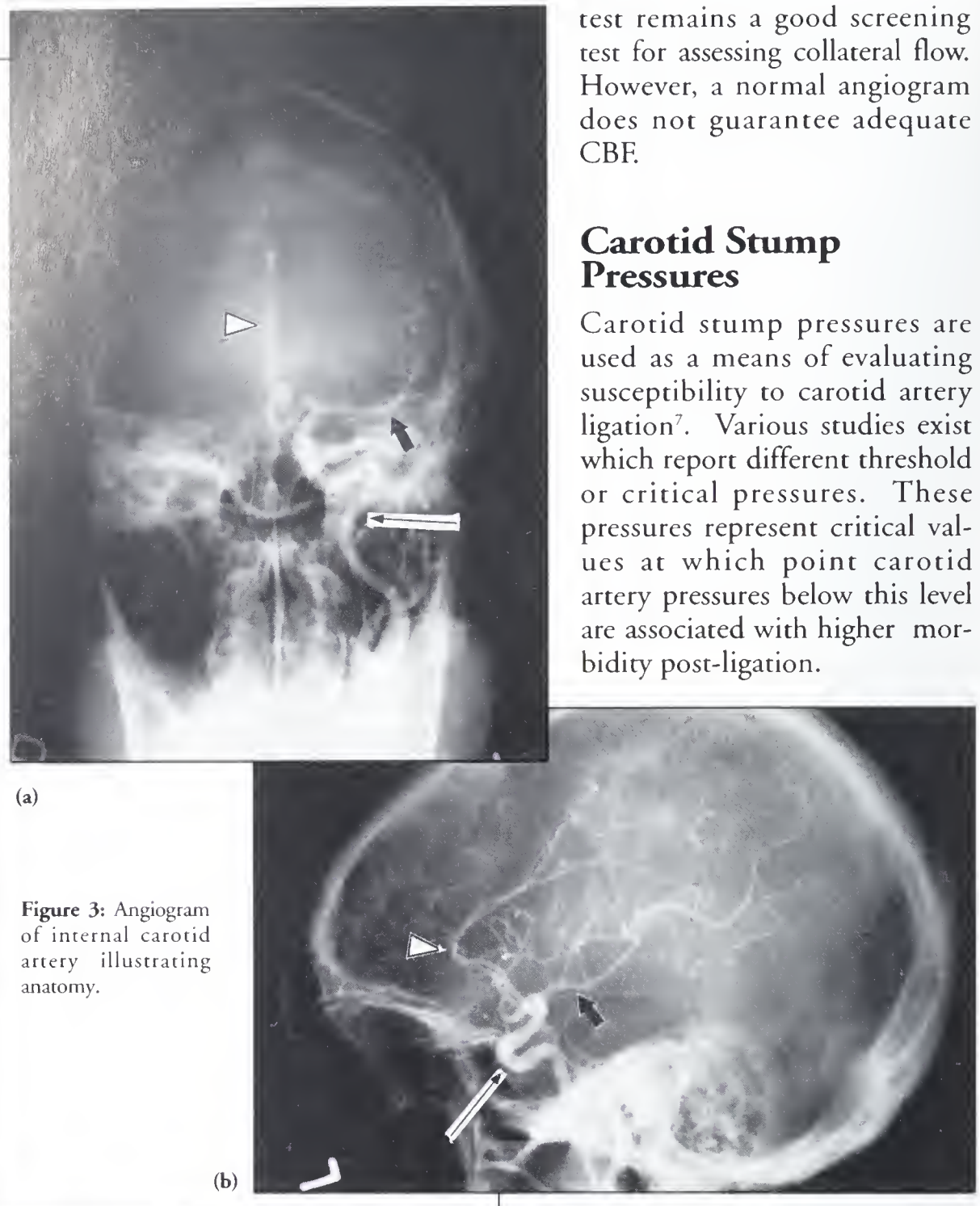
The present day version of the Matas Test calls for manual external compression of the carotid artery while observing for signs and symptoms of neurovascular impairment. Although commonly performed, this test identifies those

persons with absolutely no cerebral flow reserve. It does not assess the degree of reserve flow or any stresses which may significantly reduce CBF. In addition, when dealing with a tumour mass that encases the carotid, complete occlusion may not be achieved. This test has been associated with a 12-20% false negative result when compared with more recent accurate means of evaluation. That is, adequate collateral circulation is assumed to exist when, in fact, cerebral blood flow is markedly diminished after permanent arterial ligation.

Cerebral Angiography

The advent of cerebral angiography by Egaz Moniz in 1927 has helped to delineate vascular perfusion of the brain[5]. The main role of angiography is to determine the specific anatomy, the anatomic patency and pathology of the cerebral vessels (Figures 3 and 4). If the contralateral carotid is stenotic for reasons of atherosclerosis or tumour, then ligation of the carotid will be poorly tolerated. This is because the remaining carotid artery is unable to provide adequate CBF secondary to its diseased state. As a result, these findings represent a contraindication to carotid artery ligation[6].

Angiography measures patency of the arteries but not the arterial flow associated with that particular vessel. This test remains a good screening test for assessing collateral flow. However, a normal angiogram does not guarantee adequate CBF.



(a)

(b)

Figure 3: Angiogram of internal carotid artery illustrating anatomy.

Carotid Stump Pressures

Carotid stump pressures are used as a means of evaluating susceptibility to carotid artery ligation[7]. Various studies exist which report different threshold or critical pressures. These pressures represent critical values at which point carotid artery pressures below this level are associated with higher morbidity post-ligation.

Distal carotid pressures are measured using a double lumen catheter placed in the internal carotid artery. The outer balloon is inflated, occluding the artery, while the inner balloon is a pressure transducer, used to measure pressures distal to the occlusion. Ehrenfeld[8], in a study of 24 patients undergoing internal carotid ligation described safe stump pressures as those exceeding 70 mmHg (100% of patients (13/13) had no evidence of morbidity). Ligation with pressures less than 55 mmHg were associated with increased morbidity with 66% (2/3) of patients having had cerebrovascular complications. A carotid stump pressure range of 55-68 mmHg was associated with a notable risk of 50% (4/8) morbidity. However, other studies have suggested adequate pressures of 50 mmHg with little to no morbidity[9]. During De Vries'[10] analysis of Xenon CT, on average, higher stump pressures were associated with decreased morbidity. However, no one measurement in any individual could be associated with a predictable outcome.

Figure 4: Angiogram of vertebrobasilar arterial system.

Theoretically this test also poses other dilemmas. For instance, since distal pressure does not take into account the CBF, no accurate determination of flow can be made from pressure. If pathology exists in the MCA then flow may be reduced to a focal area of the brain, however, measured stump pressure in the carotid artery may not be affected. The converse also holds true if intact collateral flow exists but a stenosis is present just distal to the pressure transducer. In this instance, the transducer may register a falsely low pressure due to the proximal stenosis, when in fact, adequate perfusion is obtained distally from contralateral flow.

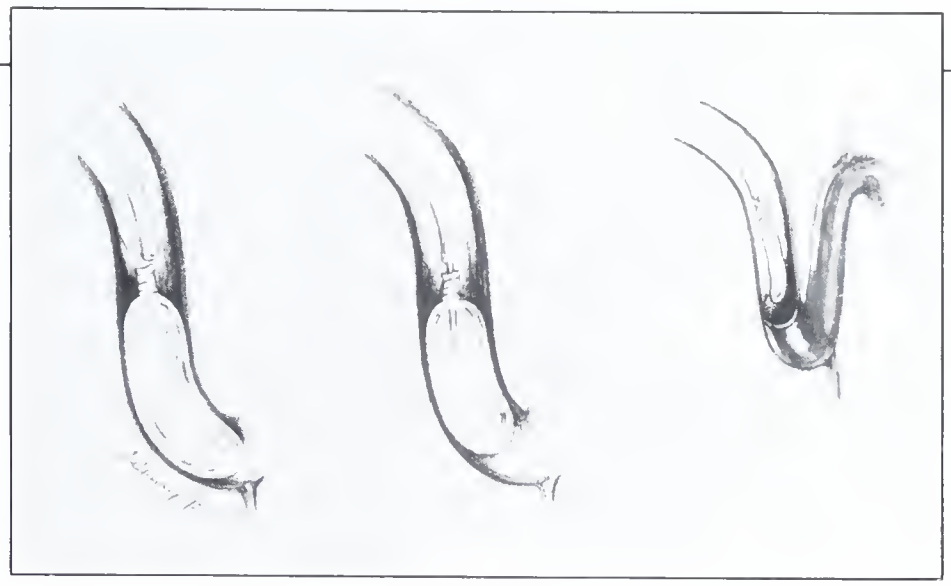


Figure 5: Balloon occlusion of the carotid artery.

Temporary Balloon Occlusion (TBO)

The early 1970's brought about the advent of a new adjunctive technique to assess the morbidity of carotid artery ligation. This method entailed the use of a non-detachable balloon used to occlude the carotid artery. The procedure of balloon occlusion was first used as a means of treating surgically inaccessible cerebral vascular aneurysms[11]. The method was later modified by the use of a nondetachable balloon. In assessing the carotid artery, the balloon is introduced via the femoral artery and catheterized rostrally towards the carotid artery. When inflated, the non-detachable balloon completely occludes the lumen of the vessel (Figure 5). This procedure essentially simulates carotid artery ligation without causing any permanent morbidity. Balloon occlusion is used in conjunction with a number of other ancillary tests such as electroencephalography (EEG), somatosensory evoked potentials (SSEP) and Xenon enhanced Computer Tomography (XeCT). Balloon occlusion eliminates the uncertainty of manual external compression of the carotid artery. The balloon remains inflated for a period of thirty minutes, during this time, neurological testing is performed to assess cerebral collateral flow. If poorly tolerated, the balloon is deflated and the catheter removed. The presence of any neurological signs or symptoms indicates poor cerebral perfusion and is deemed a positive occlusion test suggesting that the artery cannot be safely ligated or sacrificed[12].

Oculoplethysmography (OPG)

The principle of this test is based on a similar idea to carotid stump pressures. Normal retinal artery flow is associated with fine ocular pulsations. When intraocular pressure is raised above the mean systolic pressure in the ophthalmic artery, the blood flow to the eye via the retinal artery ceases as do the fine pulsations of the eye. This increased intraocular pressure may be applied to the eye by using a vacuum on the sclera. A direct relationship exists between the vacuum pressure applied and the intraocular pressure measured. When the systolic pressure just exceeds the intraocular pressure, pulsations of the eye are recorded. This test can be accompanied by compression of the ipsilateral carotid artery. In this way, blood flow will be generated by the contralateral carotid and hence collateral flow can be determined. Since the principle is based on carotid stump pressures, the same threshold value is used. In a study by Martinez et al[13], 11 patients were evaluated for carotid resection. Nine patients were determined to have adequate collateral flow. Of these patients four underwent carotid resection, none exhibiting post-operative morbidity. No patients developed any neurological sequelae. A pressure recording of greater than 50 mmHg is associated with a good prognosis. However, as with carotid stump pressures, this test is not without fault. The greatest problem is that if pathology exists in the ophthalmic artery, this will be reflected in the outcome of the test. If stenosis exists in the proximal ophthalmic artery then a low pressure will be recorded. The carotid may be functional and still provide adequate CBF in other areas. Alternatively, pathology may exist elsewhere and the OPG may be interpreted as normal.

Electroencephalogram (EEG) and Evoked Potentials (EP)

The use of both EEG and EP are used in conjunction with balloon occlusion of the ipsilateral carotid artery. Values for both tests are measured during the occlusion of the artery. These two methods rely on the same principle for their findings. Neural tissue when stimulated produces electrical output[14]. This electrical discharge is measured via surface electrodes placed on the skin.

Electrodes are responsible for detecting the post-synaptic summation potentials. Since the activity of neural tissue varies depending on the site, values recorded are proportional to the distance of electrode separation. The farther apart the electrodes, the greater the difference recorded. Results are monitored by continuous printouts monitoring both amplitude and frequency of the waveform.

Slow wave patterns are termed delta or theta waves, while fast wave forms are known as alpha or beta waves. During an episode of cerebral ischemia both the amplitude and the frequency of a wave form are known to decrease. Although noninvasive, the results of EEG testing are less than optimal. Sundt et al[15] found that EEG changes were evident only after flow decreased below 15ml/min/100g. These results have been verified by Morewetz et al[16] in their study of 127 patients undergoing carotid occlusion. In their study Morewetz found no significant changes in baseline EEG unless regional CBF (rCBF) was significantly decreased. Of the 77 patients with CBF values below 16ml/min/100g, only 5% (4/77) showed any changes in EEG. Forty two patients were found to have CBF below this value with only 21 patients showing EEG changes. Eight patients had CBF less than 9ml/min/100g and 88% (7/8) showed ischemic changes. In a study by Miller and Jawad[17] there was no correlation between severity of EEG changes and CBF. As well, there was a poor correlation with stump pressures. This method only identifies those persons at very high risk. People with low CBF reserve would not be identified. In addition, there is no way of quantifying the results found.

Evoked potentials (EP) are similar in nature to EEG. As opposed to recording neural activity the EP records the latency in a neural response. If the median nerve is stimulated the afferent nerve impulse travels from the median nerve to the spinal cord and eventually to the somatosensory area. Each time the impulse crosses a synaptic junction there is a time lag or latency. It is this latency period which is measured in EPs. It has been shown that in cerebral ischemia this latency period increases. Problems with this test are similar to those associated with the EEG.

Transcranial Doppler (TCD)

The use of TCD provides a noninvasive means to evaluate the cranial collateral circulation[18,19]. Doppler studies are based on the premise that signals (from the ultrasound) are reflected off moving objects (eg. erythrocytes). The frequency detected is in direct proportion to the velocity of the moving object. Transcranial doppler employs low frequency (1-2MHz) soundwaves to evaluate intracranial vasculature. The probe is placed on "windows" in the skull; these areas represent thinner bone areas in the skull. The commonest area evaluated is known as the "temporal window". This area is bounded by the zygomatic arch superiorly and the ear and the orbit posteriorly and anteriorly respectively. The "temporal window" gives information concerning the middle cerebral artery, distal branches of the internal carotid and proximal branches of the anterior and posterior cerebral arteries. Other windows used include the orbital as well as the suboccipital regions. TCD in these areas give rise to information regarding the ophthalmic and vertebrobasilar arterial systems respectively. TCD provides information regarding flow through specific arteries rather than regional cerebral blood flow. Results of the study are often influenced by physiologic factors such as age, end-tidal CO2, blood pressure and hematocrit.

This procedure is ideally suited for persons presenting with cerebrovascular disease. The test provides a more physiologic picture of flow in a specific artery rather than regional CBF. Giller[20] reported a case study with the use of TCD to gradually occlude the carotid artery in a lady who clinically could not tolerate sudden occlusion of the carotid artery. With use of a Crutchfield clamp and the TCD, slow closure of the clamp was completed over a period of ten days. The MCA flow velocity was recorded with every turn of the screw and clinical evaluation was undertaken. Permanent occlusion and subsequent ligation occurred 10 days later. Schneider21 in a study of 50 patients used TCD to evaluate the collateral flow before undergoing carotid endarterectomy. TCD detected collateral circulation with a sensitivity of 89% and a specificity of 80%. Patients were divided into 3 groups based on TCD results. Group I patients had good collateral flow. Group II had collateral flow but there was evidence of greater than 80% proximal occlusion. Group III had no evidence of collateral flow. During endarterectomy, EEG evaluation was also performed. In 7 patients, EEG changes were noted intraoperatively. Six of these had poor preoperative TCD results (Group II-3;Group III-3). It was also noted that Group I on average had higher stump pressures. Patients were able to be qualitatively grouped according to their risk category.

One significant drawback of TCD is that like many ultrasound interventions, the quality of the study often depends on the knowledge, skill and experience of both the technician and the radiologist. It has been found that about 5-15% of individuals do not possess adequate temporal windows and this proportion increases with age. The advantages of this technique are its noninvasive nature and the extreme portability and inexpensiveness of the machinery.

Xenon Enhanced Computed Tomography (Xe/CT)

This method involves use of stable Xenon to assess regional CBF by use of CT. Patients were catheterized with a Swan-Ganz catheter via the femoral artery. Advancement of the catheter proceeds to the internal carotid artery. A standard neurological exam is then performed both pre and post-balloon occlusion. If neurological deficits exist immediately post-occlussion, the procedure is terminated. If no sequelae are observed after 15min then the patient is transported to the CT room with the catheter deflated, but still intact. Once in the scanner, the patient is given a mixture of Xenon and air. This continues until the blood is saturated with Xenon. The balloon is inflated and a scan is obtained at 3 separate levels of the brain. After this, the catheter is removed, and a baseline CT scan is obtained about 20 minutes after removal of the catheter[22].

The method of XeCT has been extensively studied and appears to be far superior to the use of angiographic evidence to support low cerebral perfusion[23]. Yonas et al[23] selected patients for surgical cerebral flow augmentation on the basis of XeCT results. All patients presented with neurological disease. Of the 25 patients studied, XeCT identified focal hypoperfusion in eight patients. All underwent carotid bypass and have shown post-operative increases in flow. The other seventeen patients received no treatment and have done well in their follow-up with no neurological sequelae indicating good initial CBF .

This particular method provides several advantages over previously mentioned tests. Several studies have been cited with optimal results. De Vries et al[24] in a study of 136 patients found 11% who showed no clinical sequelae after temporary balloon occlusion, but had evidence of ipsilateral hypoperfusion documented by Xenon CT. This compares with earlier studies by the same author involving 4% with poor documented flow. Erba[25], in a similar study detected 44% with clinical pathology which suggested possible hypoperfusion. However, none of these patients developed clinical evidence of ischemia. Significantly, follow-up revealed that the patient undergoing ligation with XeCT evidence of hypoperfusion suffered a stroke after surgical resection. The two patients who had no XeCT changes or who had evidence of symmetric hypoperfusion had no neurological morbidity postsurgical resection. It seems quite evident that clinical evaluation alone misses a significant number of patients. This was further evident in a previous study done at this institution[26]. In this study, 29 of 30 (97%) patients were found to have adequate cerebral circulation as determined by test balloon occlusion with clinical surveillance. Of these, 55% (16/29) went on to some form of carotid artery sacrifice. Five (31%) suffered neurological complications while the mortality rate was 13% (2/16). The XeCT CBF studies also confirmed several findings with other tests.

De Vries et al[24] also confirmed that although associated on average with a higher pressure, no single carotid stump pressure could be used as a predictor of success of carotid artery ligation. Furthermore, the XeCT method can also be used to derive quantitative results of CBF. As previously mentioned this may be more important than qualitative reporting.

There are several disadvantages of this technique. The first is that the patient must be transferred from the angiographic suite to the CT room with the catheter in situ. This requires extensive personnel and the catheter must be inflated without the use of angiographic aid. This could theoretically cause problems with dislodgement or with re-inflation. To date, no such cases have been reported. Many institutions would have to fit the CT scanner with the ability to detect Xenon. This may not prove economical for all institutions. Finally, the present systems available only allow 3 different levels of imaging. Thus, focal areas of hypoperfusion could again theoretically be neglected. Studies by Gur[27] have demonstrated that Xenon has a propensity to increases CBF. This occurs on a global scale rather than being focal or tissue specific. Values used were slightly higher than those used in clinical studies but within the same magnitude. As the effect was consistent this should not detract from the results obtained by XeCT.

^{99m}Tc-Hexamethylpropyleneamine Oxime Singel-Photon Emission CT (^{99m}Tc-HMPAO SPECT)

The use of single photon emission computed tomography has recently been evaluated[28,29,30]. In addition, the use of ^{99m}Tc hexamethylpropyleneamine oxime (^{99m}Tc-HMPAO) has been employed as an intravenous contrast agent. Studies involve first assessing the patient with balloon occlusion for any neurological findings; if asymptomatic further testing is done. Intravenous ^{99m}Tc-HMPAO is injected while the ipsilateral carotid is occluded. The balloon is left inflated for a period of five minutes. The contrast agent is lipophilic and taken up by the cells after traversing the blood brain barrier. Once in the cells, the ^{99m}Tc-HMPAO is rapidly converted to a hydrophillic substance. The patient then undergoes SPECT scanning to determine areas of hypoperfusion (Figure 6,7). This scan is usually compared to a baseline scan taken 24 to 48 hours prior to balloon occlusion. Although small numbers were used in both studies, initial investigation suggests a good correlation between scan results and post-occlusion outcome. If no significant alteration is noted between baseline and occlusion studies, then the patient is considered a good risk candidate for not developing occlusive neurological sequelae. However, the converse remains unproven. That is, if asymmetry exists between the two scans then the patient may or may not be able to tolerate internal carotid occlusion. This eludes to one of the major disadvantages of SPECT scanning, that is, its inability to determine absolute

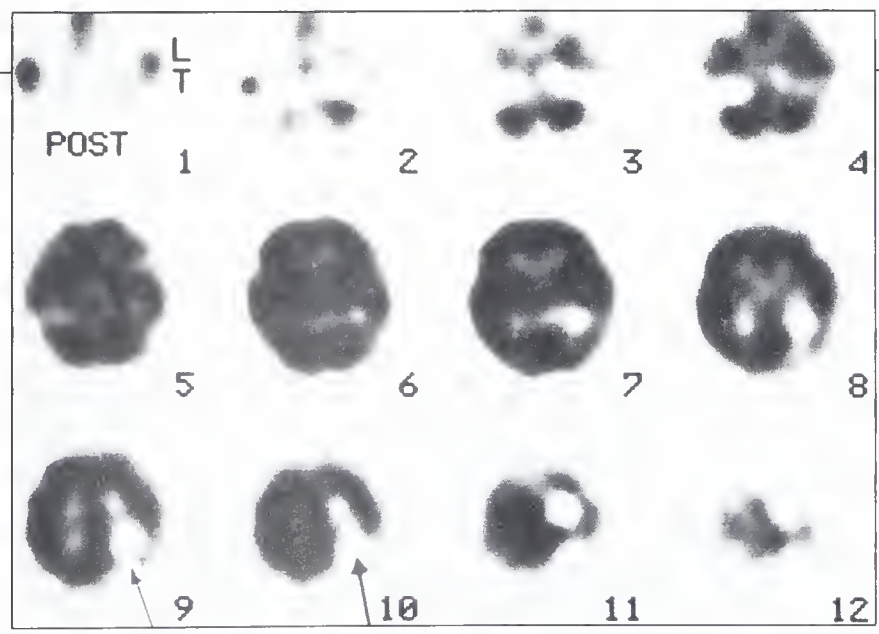


Figure 6, 7: Nuclear imaging (^{99m}Tc-HMPAO SPECT) delineating areas of hypoperfusion.

quantification of CBF. Monsein et al[28] have used an equation to indirectly calculate CBF but the task remains cumbersome and has not been well validated. This remains an important consideration as one patient in the study had relative hypoperfusion post-occlusion. The absolute quantification of CBF revealed greater than 20ml/min/100g providing adequate flow. The patient tolerated permanent occlusion without neurological sequelae. Thus, if determined by scan alone this patient would have been overlooked. In addition, 36% (4/11) of patients in the study who had changes on SPECT all had CBF values greater than 40ml/100g/min. The study revealed that 5 of 11 patients (45%) although neurologically stable, demonstrated changes on SPECT scanning. Similarly, Peterman et al[29] showed 2 of 17 (12%) patients having SPECT changes present without suffering compromised CBF. The main advantage of this assessment includes the readily available access to the equipment and the lack of need to transport patients with a catheter in the carotid.

Conclusion

The propensity for head and neck cancer to invade surrounding tissue is great. The surgeon must be increasingly aggressive if surgical cure is to be sought. Ligation of the carotid artery has long been an impediment to the complete resection of tumours. With recent advances in technology we are able to better predict outcome preoperatively. In particular, it appears that clinical assessment alone of cerebral blood flow during test occlusion of the carotid artery will miss a great proportion of patients. Carotid artery ligation in these people will often be associated with a failure rate of 15-20%. At this institution we advocate the use of TBO with XeCT to appropriately assess CBF prior to carotid artery ligation (Table 1). Patients with reduced CBF can be identified with the use of XeCT providing a more valid adjunct to assess collateral circulation.

Table 1

Pre-occlusion Test	Investigator	Sensitivity
OPG	Martinez[13]	++
EEG	Miller[17]	+
Carotid Stump Press.	Ehrenfeld[8]	++
XeCT	De Vries[22,24]	+++
TBO c hypotension	McIvor[26]	+++

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ERRATA

The author of the "Annual Medical Student Research Day" article in the May 1994: Vol 71, No. 3 issue of the UTMJ was incorrectly stated in the table of contents on page 3 as D'Arcy Little.
It should have read Dr. Catherine Whiteside.

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Dysphagia: Approach to Diagnosis and Management

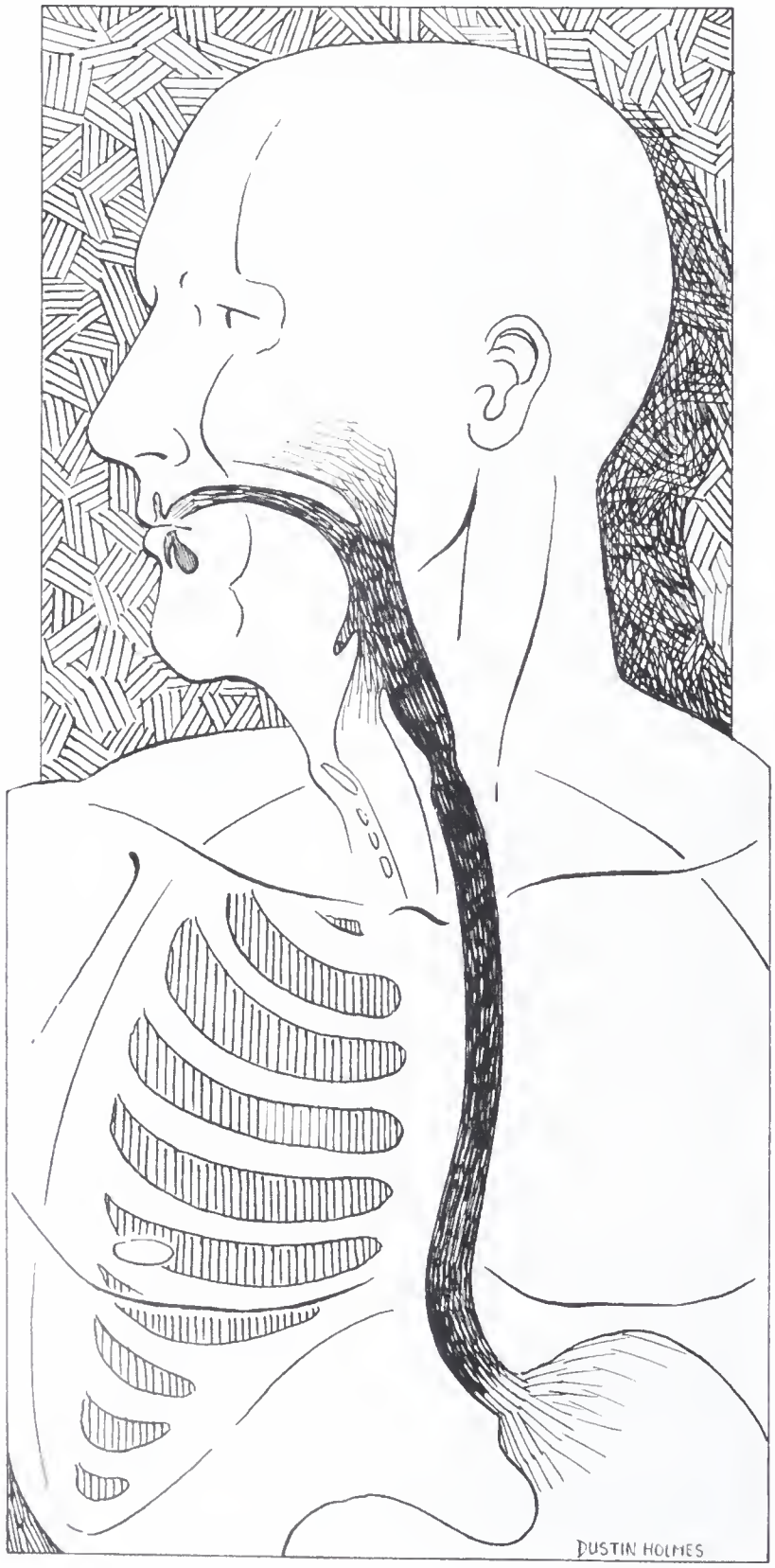
Nick F. Logarakis (97 5)

Abstract

Dysphagia is the subjective awareness of difficulty in swallowing. The mechanism of swallowing is a highly integrated process, and the approach to managing patients with dysphagia should focus on the pathophysiologic cause. The work-up should include a complete history and physical examination, along with various imaging techniques to localize the defect to one of the stages of swallowing (oropharyngeal or esophageal), and to characterize the cause as a structural or a motility disorder. Management should focus on relieving symptoms, maintaining function, and preventing complications. A team approach with multispecialty input is important for optimal management.

L'abrégé

La dysphagie est la conscience subjective de la difficulté à avaler. Le mécanisme de déglutition est un processus très intégré, donc l'approche de traitement des patients atteints de dysphagie doit être concentrée sur la cause pathophysiologique. L'étude du cas doit comprendre un passé médical complet ainsi qu' un examen médical accompagné de plusieurs techniques d' images pour localiser l' anomalie qui se produit à l' un des stades de déglutition (de l'oropharynx ou de l' oesophage), et pour caractériser la cause comme un trouble structural ou comme un trouble de motilité. Le traitement doit être concentré sur le soulagement des symptômes, soutenir la fonction et prévenir les complications. Une collaboration d' équipe avec la contribution de nombreuses spécialités médicales est importante pour un traitement optimal.



DUSTIN HOLMES

Introduction

Swallowing is a complex process that begins with propulsion of a bolus from the mouth to the pharynx, and is completed by its passage through the lower esophageal sphincter(LES) into the stomach[1]. Simultaneously, during swallowing the airway is protected from inappropriate substances. Dysregulation results in aspiration and malnutrition, the main causes for concern in swallowing disorders[2].

Dysphagia is the subjective awareness of difficulty during the act of swallowing. Hendrix (1993) reports that one survey found dysphagia to be present in 12% of admissions to a general hospital[3]. Siebens et al.(1986) state that as many as 50% of nursing home residents have swallowing dysfunction[4]. Patient complaints about problems during swallowing should be investigated, and the

purpose of this paper is to provide an approach to diagnosing and managing patients with dysphagia.

Dysphagia may be divided into two types: oropharyngeal and esophageal dysphagia. In oropharyngeal dysphagia there is difficulty in initiating deglutition. Esophageal dysphagia is characterized by the inability to maintain transit through the esophagus[5]. Dysphagia as a result of esophageal disease may be either a structural or motor abnormality. Patients commonly complain that "food sticks" or "slows down" during its passage from the mouth into the stomach[5,6,7]. By contrast, with pharyngeal muscle weakness, neuromuscular dysfunction, or central/peripheral nervous system disease, the patient may complain of difficulty initiating a swallow or difficulty in passing the bolus into the esophagus. Dysphagia may occur only with a solid bolus of food, only with liquids, or in some situations with both solid food and liquids[6]. Dysphagia for liquids is characteristic of motor disorders, whereas swallowing difficulty for solid food suggests a mechanical obstructing lesion[6].

A complete, careful history and physical examination are crucial before various imaging techniques are used to confirm the specific cause. Management planning relies on an accurate and complete dysphagia diagnosis. A team approach with multi-specialty input is important for optimal management.

Physiology of Swallowing

The central and peripheral nervous systems control swallowing. Swallowing is a complex, integrative, coordinated process involving peripheral sensory inputs, cerebral cortex, brainstem nuclei, cranial nerves, cervical spinal nerves, and muscle activation.

The oropharyngeal muscles and upper-third of the esophagus and sphincter are striated, whereas the middle third of the esophagus is composed of both striated and smooth muscle, and the lower third of the esophagus including the LES is composed of smooth muscle. During swallowing, the bolus to be swallowed is propelled into the pharynx by placement of the tongue on the roof of the hard palate. Contraction of the upper pharyngeal constrictors and movement of the soft palate separate the oropharynx from the nasopharynx. With deglutition, the upper esophageal sphincter relaxes with contraction of the middle and lower pharyngeal constrictors, and relaxation of the cricopharyngeal muscle.The trachea is sealed off by the downward movement of the epiglottis and the upward movement of the glottis. Bolus transfer into the esophagus is provided by lingual propulsion, laryngeal elevation, and gravity[8,9]. Peristaltic contraction due to sequential activation of the muscles of the pharynx and esophagus by neural impulses from the "swallowing centre" propel the bolus into the stomach. The high resting pressure of the upper esophageal sphincter is re-established, preventing reflux of material from the esophagus. The peristaltic wave causes the LES to contract and re-establish its resting tone to prevent gastroesophageal reflux[3].

Cortical mechanisms play a critical role in the initiation and regulation of swallowing[10]. Multiple discrete regions of the central nervous system, when stimulated, can elicit pharyngeal swallowing. These include a specific site of the cortex, parts of the limbic pathway, and brainstem regions[11]. It was suggested that the critical site of the central swallowing pathway was in a region of the reticular formation in the area of the upper medulla and lower pons because when damaged, peripheral nerve stimulation could not evoke the pharyngeal motor pattern[12]. Other studies suggest that central control exists in two regions: one in the dorsal region of the brainstem around the nucleus tractus solitarius, and a ventral region around the nucleus ambiguus. The dorsal region elicits pharyngeal swallowing and can evoke both pharyngeal and esophageal swallowing with representation on each side of the medulla. The ventral region organizes the sequential activation of motor nuclei groups[13].

Pharyngeal and esophageal swallowing is triggered by sensory feedback from mucosal receptors to touch, pressure, chemicals, and water in the pharynx and larynx[13]. Thermal sensory input(s) to the lateral sensorimotor cortex may also influence swallowing[10]. Cortical electrical stimulation of overlapping regions of the anterolateral frontal, lateral pericentral, and insular cortex may evoke swallowing along with mastication, sucking, salivation, and gustation[10]. Oropharyngeal mechanosensory inputs to the primary motor cortex representing the tongue may be utilized in the initiation and perhaps the guidance of swallowing, although the functional organization of the cortex in relation to swallowing needs to be investigated further[10].

Studies suggest that outputs from the cortical swallowing area are relayed by interneurons within the solitary tract nucleus and reticular formation[10]. That motor nuclei innervating the muscles active in swallowing are driven and controlled by interneurons is demonstrated by studies where stimulation of the motor nuclei of the pharyngeal phase muscles does not evoke pharyngeal swallowing but only contraction of the innervated muscles[11].

Pathophysiology

Dysphagia results from an abnormality in one or more phases of the swallowing process. Dysphagia can be divided into oropharyngeal and esophageal dysphagia.

Oropharyngeal dysphagia is characterized by difficulty in initiating a swallow. Onset of dysphagia may be sudden (eg. cerebrovascular disease) or slowly progressive (eg. degenerative or demyelinating diseases)[3]. Patients may present with the following symptoms: food sticking in the middle of the neck[6,14]; nasal or oral regurgitation of liquid or food after swallowing[6,14]; cough/choking due to aspiration[6,14]; lip weakness and drooling of saliva[3]; speech disorders(eg. wet sounding voice during or after feeding)[3,10]; and pneumonia with or without a history of dysphagia[3].

Table 1: Etiology of Oropharyngeal Dysphagia

Neurologic Disease

Central Nervous System
Parkinson's disease
cerebrovascular disease
brainstem tumour
spinocerebellar degeneration
amyotrophic lateral sclerosis
multiple sclerosis
Alzheimer's disease
Huntington's chorea
bulbar poliomyelitis
tabes dorsalis
Creutzfeldt-Jakob disease
head trauma

Peripheral Nervous System
peripheral neuropathy (ie. DM)
Guillain-Barre syndrome

Motor End Plate
myasthenia gravis
myasthenic(Lambert-Eaton) syndrome

Skeletal Muscle
polymyositis
dermatomyositis
muscular dystrophy
alcoholic myopathy
thyroid associated myopathy
oculopharyngeal syndrome
systemic lupus erythematosis
collagen vascular disease
congenital myopathy
diffuse idiopathic skeletal hyperostosis

Local Structural Lesions
oropharyngeal cancer, trauma
surgical resection
Zenker's diverticulum
cervical spurs
thyromegaly
infections (ie. pharyngitis)
pharyngeal/laryngeal paralysis
Ludwig's angina
oral ulcers (ie. herpes, aphthous)

(Adapted from[3,4,6,14])

The etiology of oropharyngeal dysphagia is presented in Table 1. Neurologic disease accounts for 80% of cases of dysphagia, and the rest are usually due to local structural lesions[6,14]. Myasthenia gravis, dermatomyositis, and thyroid myopathy should be considered in the differential diagnosis since each has a specific and effective treatment that may alleviate the symptoms[3]. Hemispheric cerebrovascular disease or cerebral palsy may result in deficits of the oral, pharyngeal, and esophageal stages of swallowing as a result of damage to corticobulbar output pathways and possibly from damage to the neural pathways involved with the processing of afferent inputs from the oral cavity, upper respiratory tract and esophagus, which are stimulated by the bolus[10].

Esophageal dysphagia may be due to an abnormality where the esophagus is not able to move solids (mechanical dysphagia) or solids and liquids (motility dysphagia)[6]. It is more common than oropharyngeal dysphagia[6,14]. Symptoms occur either retrosternally or at the level of the suprasternal notch[14]. Refer to Table 2 for the etiology of esophageal dysphagia. Any extrinsic or intrinsic structural lesion may impede bolus transfer down the esophagus. The commonest mechanical lesions are peptic stricture, esophageal cancer, and a lower esophageal (Schatzki) ring[6,14]. Heartburn and regurgitation of sour/bitter material is characteristic of gastroesophageal reflux disease. Motility disorders of the esophagus are less frequent than mechanical lesions[14]. Refer to Table 2 for the etiology of esophageal dysphagia. Motility disorders that delay esophageal transit time commonly include achalasia, diffuse esophageal spasm, and nonspecific motor dysfunction[6,14]. Nonspecific motor dysfunction is a category of disorders including vigorous achalasia, nutcracker esophagus, and hypertensive LES, that do not meet the criteria for achalasia and diffuse esophageal spasm.

Decktor et al. (1990) studied the relationship of esophageal dysphagia and heartburn. They evaluated 1035 dysphagic patients, with 762 (74%) also complaining of heartburn. Interestingly, no pattern diagnostic of a specific esophageal motility disorder was observed. Normal motility was present in 40% of patients with heartburn, compared with 27% of patients without heartburn[5]. Odynophagia, painful swallowing, frequently indicates severe mucosal inflammation. In the retrosternal region, esophagitis due to Candida, Herpes, or CMV infections should be suspected, especially in immunocompromised patients[1]. Odynophagia may also be due to any condition that alters the peristaltic activity of the body of the esophagus[6].

Drug-induced dysphagia can be due to a normal drug side effect, a complication of the therapeutic action of the drug, or a medication-induced esophagitis. Many drugs and hormones cause gastroesophageal reflux disease by decreasing the LES pressure[15]. Dysphagia may be caused by drug-induced xerostomia. Decreased saliva in the mouth impairs oropharyngeal bolus transport, reduces acid neutralization in the distal esophagus after gastroesophageal reflux, and increases the likelihood of the drug adhering to the esophageal mucosa producing injury[15]. Xerostomia is also associated with Sjogren's syndrome.

Psychological factors may aggravate, but rarely cause dysphagia[4]. Swallowing problems in psychiatric patients must

Table 2: Etiology of Esophageal Dysphagia

Mechanical Disorders

Intrinsic
esophageal cancer
benign tumours
peptic stricture
strictures due to caustic ingestion
lower esophageal (Schatzki) ring
esophageal webs
foreign bodies
lymphoma

Extrinsic
vascular compression (ie. dysphagia lusoria, dysphagia aortica)
mediastinal abnormality (ie. substernal thyroid, enlarged lymph nodes)
paraesophageal hiatus hernia

Motility Disorders
achalasia
diffuse esophageal spasm
scleroderma
nonspecific esophageal motor dysfunction
presbyesophagus
Chagas' disease
amyloidosis
diabetic neuropathy
drugs (ie. anticholinergic)

(Adapted from[6,14])

be evaluated since many antipsychotic drugs produce swallowing dysfunction as a side effect[3]. Globus hystericus is a benign condition where patients sense a lump in the throat. They may complain of dysphagia, but swallowing is normal. Generally, it is due to the tightening of the upper esophageal sphincter.

Diagnostic Approach

History and Physical Examination

By taking a careful, complete history one can localize the defect to one of the three stages of swallowing (oral, pharyngeal, or esophageal) and characterize the responsible disorder as a structural (anatomic) or motility disorder in the majority of patients[1,3]. Important information in the history includes: the types of foods (solid and/or liquid) causing dysphagia; acute or progressive onset; duration; its localization; continuous or intermittent symptoms; relieving factors; aggravating factors; current feeding status; and associated symptoms such as heartburn, regurgitation, odynophagia, cough/choking, weight loss, chest pain, and voice changes. Refer to Figure 1 for a diagnostic algorithm for dysphagia. A drug history is required because many commonly used drugs affect salivary flow. Dysphagia should never be dismissed as a psychological manifestation[3].

A few months' duration of worsening dysphagia suggests progressive mechanical narrowing of the lumen, as may occur with an esophageal carcinoma. Benign disease is symptomatic for longer periods of time[16]. The patient's diet may change from solids to pureed foods and liquids as the symptoms worsen[3]. Dysphagia with liquids suggests a neuromuscular motility disorder[17]. Patients with esophageal spasm may have dysphagia and odynophagia, especially with extremely cold or hot liquids[16]. Ingestion of caustic agents (eg. lye) suggests an esophageal stricture secondary to severe chemical esophagitis[16]. Cough and/or choking with swallowing is suggestive of pharyngeal dysphagia due to a neurologic disorder[3]. Localization of structural abnormalities causing dysphagia in the pharynx is accurate. In the esophagus, localization is to the throat, leading to the false assumption that the problem is pharyngeal dysphagia[3].

The physical examination should include a careful examination of the mouth, pharynx, hypopharynx, and larynx. A neurologic examination with cranial nerve testing should be included[4]. Observing the patient swallow may be useful in determining the timing of the symptoms. Dysphagic patients may present with malnutrition, dehydration, or pneumonia. Usually, physical findings are normal but weight loss and lymphadenopathy suggest a chronic or potentially malignant condition.

Routine laboratory tests are usually normal. A patient with chronic dysphagia and weight loss may have low serum albumin and cholesterol levels[6]. Anemia may be present as a result of bleeding[6] from an esophageal neoplasm.

Radiographic Studies

As an initial study, barium swallow radiography is performed to exclude morphologic lesions. If no abnormality is seen, an esophageal motility study using a barium-coated marshmallow to evoke symptoms should be performed[1,14]. Prolonged transit times are seen in motor disorders. Barium swallow is the commonest method of investigating structural lesions and motor disorders of the esophagus[16]. Motility disorders are best evaluated by radiographic studies[17]. Barium studies are relatively insensitive in detecting mucosal disease[16]. Radiographic studies were reported to be superior than endoscopy for diagnosing submucosal tumours, extramural tumours, achalasia and hiatus hernia[18]. The chest radiograph is usually normal, although if the esophagus is enlarged and dilated and there is an absent gastric bubble under the left diaphragm, achalasia should be suspected[6]. Aspiration pneumonia may be present in a patient with neurogenic oropharyngeal dysphagia.

MRI is more sensitive than CT in detecting small and acute cerebral infarcts[19], but a negative MRI study does not rule out brainstem stroke in a patient presenting with pharyngeal dysphagia, possibly associated with dysphonia or dysarthria[20].

Endoscopy

Endoscopy is an inexpensive, quick, safe method of screening for aspiration, pathological lesions, and swallowing dysfunction[2]. It permits direct visualization and histological assessment of structional lesions. Endoscopy with biopsy or brush cytology is indicated in most patients with esophageal dysphagia for non reflux-related esophagitis[14]. Patients with motor disorders (eg. achalasia) are occasionally endoscoped to ascertain if dilatation of the LES may be undertaken[6] and to exclude underlying pathology[14].

Gastroesophageal reflux disease produces endoscopically-detectable inflammatory changes in the mucosa of the distal esophagus, although normal findings on endoscopy cannot rule out reflux disease[21]. Submucosal or extramural lesions can be missed by endoscopy, but endosonography (EUS), by combining endoscopy with ultrasonagraphy, has proven more successful[21]. Indications for EUS include local tumour staging, detection of lymph nodes, submucosal tumours, and tumour exclusion in patients with achalasia and peptic strictures. The accuracy of determining tumour depth infiltration ranges between 79% and 92% with EUS[12]. The endoscopic detection of early asymptomatic esophageal cancer can be improved by using toluidine blue which stains tumour cells[21]. Endoscopic therapeutic techniques include foreign body removal, laser coagulation and thermocoagulation, hemostasis by injection therapy, and tumour mass reduction by laser treatment[22].

Manometry

Intraluminal pressures at multiple sites along the esophagus are recorded simultaneously during the resting state and at swallowing. Manometry is used in patients with esophageal motor disorders. Manometry combined with the acid perfusion test and/or pharmacological stimulation of the esophagus is commonly used to evoke abnormal contractions and reproduce symptoms[16]. The most commonly detected manometric findings suggest nonspecific motor disorder secondary to gastroesophageal reflux, and neurogenic motor disorders[2]. Aperistalsis is present in both esophageal scleroderma and achalasia, with an incompetent LES and hyperactive LES, respectively.

Radionuclide Scintigraphy

Gastroesophageal reflux and esophageal transit time are determined with the ingestion of a radioisotope-labelled bolus and scanning over the chest. It is used in detecting motor dysfunction, and may have a role in assessing gastroesophageal reflux disease[16].

Bernstein Test

This test is used to determine whether the patient's epigastric pain is secondary to acid reflux. This test involves intraesophageal perfusion of saline, acid, and then antacid with the patient unaware as to what is being administered. This test is considered positive when the patient's symptoms occur during acid perfusion and relieved by antacid perfusion[16].

Management Approach

Dysphagia should be approached from a pathophysiologic point of view[1]. Management requires a coordinated multidisciplinary team that may include gastroenterology, otolaryngology, neurology, radiology, speech pathology, dieticians, and nursing staff. Treatment can involve dietary manipulation, pharmacologic therapy, therapeutic endoscopy, and occasionally surgery. If nonsurgical therapies fail to improve a patient's symptoms, operative repair should be offered to good surgical candidates[22].

In an emergency department situation, in which a patient presents with an acute complaint that a bolus of food is stuck in the esophagus, an esophagogram with a small amount of barium should be done. Endoscopy should be performed to remove the lodged bolus and determine the cause of the obstruction. Esophageal rupture may occur with attempts to push the bolus into the stomach[6].

The commonest causes of esophageal dysphagia requiring surgical correction are Zenker's diverticulum, paraesophageal hiatal hernia, achalasia, and esophageal carcinoma[22]. Surgical techniques used to correct esophageal dysphagia include: 1) removal of the obstructing lesion endoscopically; 2) correcting any abnormal condition resulting in esophageal angulation and obstruction (eg.incarcerated paraesophageal hiatus hernia); 3) perform a myotomy to weaken the esophageal musculature (eg. for achalasia); 4) resection of the obstructing segment and reconstruction with gastric or intestinal tubes[22].

In the chronic setting, nutritional status monitoring through weekly weighing or caloric counts is essential[23]. Maintaining safe oral feeding is the goal of treatment for the dysphagic patient. When it becomes unsafe or fails to provide adequate nutrition other feeding methods must be used[23]. Nasogastric feeding is adequate for short-term (3 to 6 months) nutritional support[23]. Swallowing complications of NG tubes are reduced by using a small-bore nasoduodenal tube with radiologic confirmation of its position[2]. Patients with"intractable" dysphagia, especially those who have had surgical resections involving the mouth and pharynx, may require a gastrostomy or percutaneous gastrostomy tube[24]. Enteral feeding does not eliminate the risk of the patient developing aspiration pneumonia[23]. If aspiration of large volumes of saliva is present, management options include reducing salivary flow (eg. atropine, hyoscine), suctioning through a minitracheostomy, discontinuing oral feeding, or surgery in patients who do not respond[2].

Drug-induced dysphagia may be improved by removing the causitive agent, substituting an alternative drug, increasing fluid intake with drug administration, sitting in an upright position, reducing pill size, and avoiding medications at bedtime[15].

Dietary modifications may help reduce dysphagia. The amount and rate of intake should be controlled to prevent aspiration[23]. This may be achieved by taking small bites and sips. Thick fluids and puddings are better than clear liquids in that they maintain the form of the bolus and do not spill into the trachea easily[2,23]. Avoidance of spicy foods, and limiting intake of alcohol, coffee, tea, and cola beverages may help. While feeding, the patient should sit fully upright, with the head flexed forward and midline[23]. The head of the bed should be elevated while sleeping[4].

Management

Neoplasm

Esophageal cancers are primarily squamous cell carcinomas, and risk factors for their development include smoking, alcohol consumption, achalasia, lye-induced stricture, and head and neck cancer history. Typically, the patient is over 40 years of age, smokes, drinks alcohol, has rapidly progressive dysphagia and weight loss. Studies support that adenocarcinomas comprise between 1% and 5% of esophageal cancers[6,14]. Adenocarcinomas often arise in Barrett's esophagus (metaplasia from squamous epithelium to columnar-lined esophageal epithelium)[14]. Studies show a 2% to 10% frequency of malignant degeneration,[25,26] which is detectable by endoscopy with biopsy specimens in 89.1% of cases[21].

Treatment options include radiotherapy for carcinoma of the proximal third of the esophagus, surgical resection for the distal third if there is no evidence of metastatic spread, and a combination of surgery, radiotherapy, and chemotherapy for lesions of the middle third[6]. Surgery involves resection of the esophagus and tumour, and all distal esophagus. Subsequent reconstruction involves the use of gastric tubes or segments of transposed large or small intestine[22]. If inoperable, dysphagia relief may be achieved with esophageal dilatation, laser endoscopy, and intraluminal tubes[22].

Benign Stricture

The commonest benign stricture is peptic stricture with distal esophageal narrowing resulting from chronic gastroesophageal reflux. Typically, the patient complains of continuous and progressive dysphagia for solids, heartburn, and weight loss[14]. Radiographically, they are smoothly tapered and of variable length. Ambulatory 24-hour esophageal pH monitoring with acid exposure (ie. pH<4) of 6% or more is considered abnormal[5]. Esophageal pH monitoring also identifies patients at risk for reflux aspiration[2]. Treatment may include simple measures such as sleeping with the head of the bed elevated, avoiding bending over or wearing tight garments, and losing weight. Pharmacologic therapy may include antacids after meals and at bedtime, H2 antagonists (which decrease gastric acid production), and increasing the LES pressure with metoclopramide or bethanechol which may decrease the volume of reflux.

Caustic agent ingestion produces a long stricture in the proximal esophagus with mucosal interruption[6]. Barrett's esophagus produces benign mid or proximal strictures. Other therapies include dilatation with bougies or with endoscopically placed balloons. Surgery is done when there are extensive strictures or when dilatations have failed[6].

Mucosal Ring (Schatzki ring)

This is commonly referred to as the "steak house syndrome" because of intermittent dysphagia for solids, especially meats[6,14]. Lumenal diameter of less than 12 mm always causes dysphagia[14]. It is often associated with hiatus hernia[6]. Confirmation of the diagnosis is made by a barium swallow or administration of a 12 mm diameter barium tablet. Management includes educating the patient with respect to chewing food well, as well as esophageal dilatation.

Achalasia

In achalasia, the smooth muscle contractions in the body of the esophagus are weak and uncoordinated. There is excessive resting tone in the LES. The etiology is unknown. The esophagus is dilated, terminating in a smooth tapered beak and there is impaired passage of barium into the stomach[6,14]. Manometry helps to confirm the diagnosis. Approximately 2% to 8% of patients presenting with dysphagia have achalasia[22]. Patients have gradually progressive dysphagia for solids and liquids with frequent weight loss. Treatment options include pneumatic dilatation of the esophagogastric junction or a lower esophageal sphincterotomy[6].Other pharmacologic therapies include long-acting nitrates, calcium channel blockers, and beta-2-adrenergic agonists[22].

Diffuse Esophageal Spasm

Symptoms of diffuse esophageal spasm include intermittent dysphagia for solids and cold liquids associated with retrosternal chest pain. Chest pain radiating to the back may be mistaken for angina pectoris[14]. Cardiac investigations must also be carried out as a precaution[7]. It may be precipitated by gastroesophageal reflux or stress, and may evolve into achalasia[14]. Diagnosis may be made by manometry[6,14]. Endoscopy should be done only to rule out malignancy if it is suspected[4]. There is no definitive therapy, but patients have been treated with esophageal dilatation.

Scleroderma

In this systemic disorder, esophageal involvement is present in about 75% of patients, the majority of whom have Raynaud's phenomenon[6]. There is weak contraction and peristalsis in the distal esophagus and LES, predisposing to reflux and ensuing damage[6]. Diagnosis is made by manometry, and barium studies which reveal a dilated esophagus. Treatment includes pharmacologic therapy to prevent reflux. Strictures are treated with esophageal dilatation.

Conclusion

Dysphagia can arise from a number of etiologies. A careful history and physical examination point toward a specific diagnosis in most patients and provide the basis for the choice and order of diagnostic tests. Treatment options may include diet manipulation, pharmacologic therapy, therapeutic endoscopy, and surgery. Multi-specialty management and follow-up is essential for optimal results.

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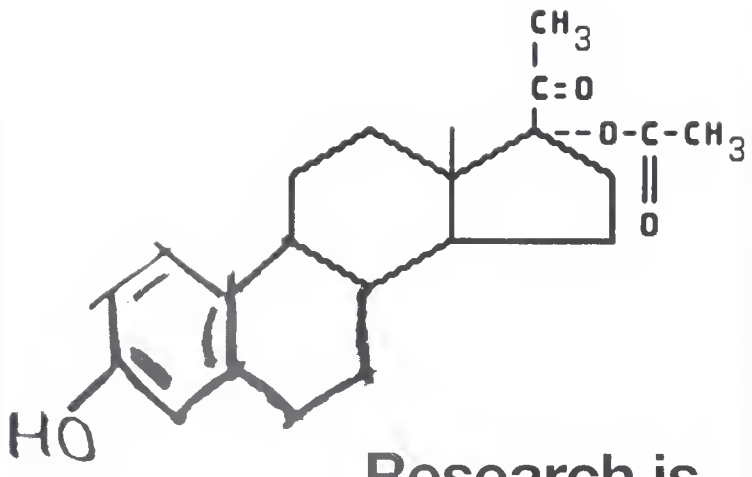

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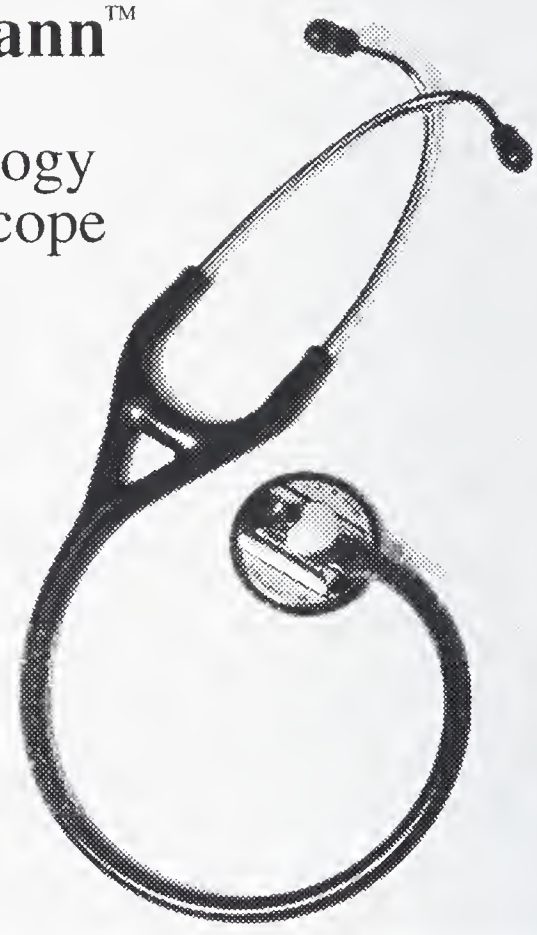

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The Medical Post

Blepharoptosis: A Clinical Approach

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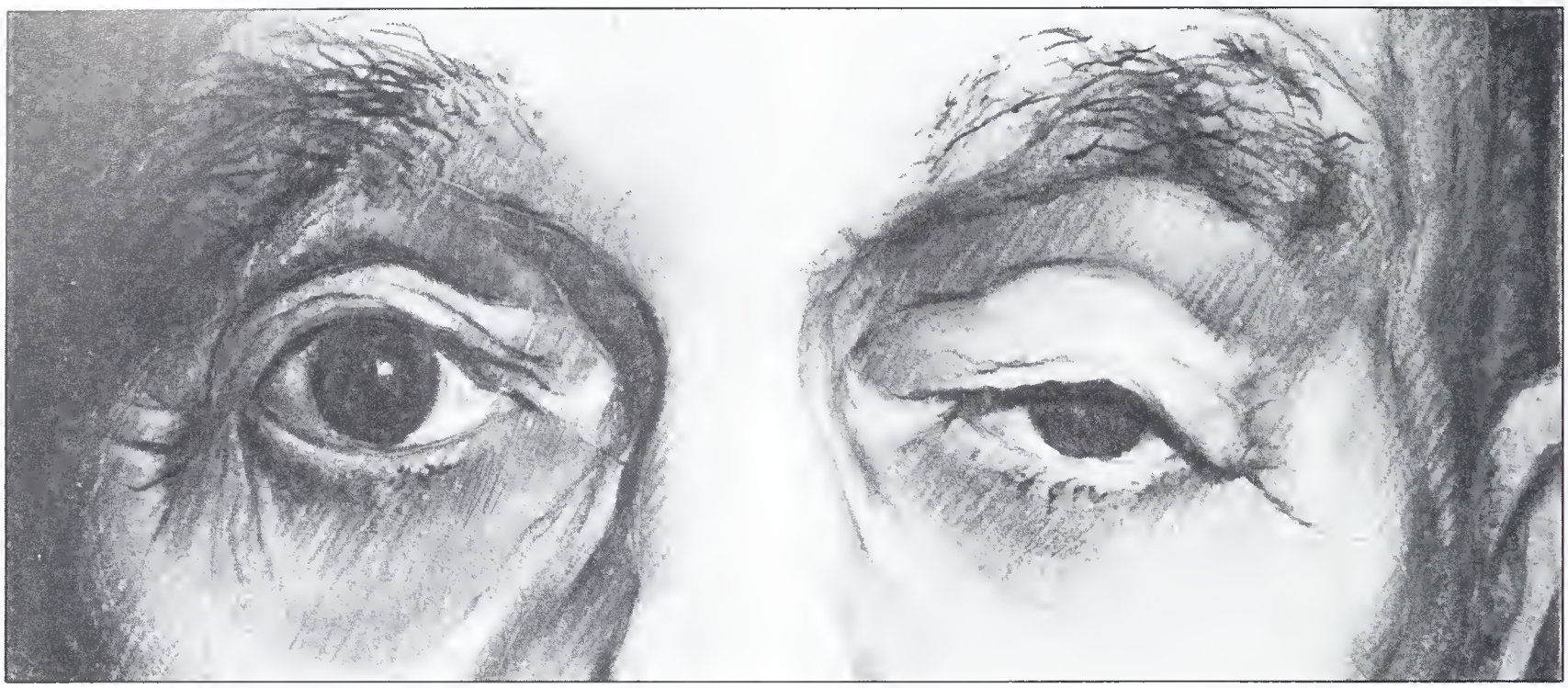

JAY OLAUSON

Abstract

Blepharoptosis (ptosis) is abnormally low position of one or both upper eyelids. This article summarizes the regional anatomy of the eyelids, and the current classification, evaluation, and differential diagnosis of ptosis. Surgical and non-surgical treatment options are described. Post-operative complications are outlined.

Introduction

Blepharoptosis (ptosis) is abnormally low position of one or both upper eyelids[1]. The normal position of the upper lid is midway between the superior limbus and the upper pupillary margin. This may vary by 2 mm provided the lids are symmetric.

Review of Eyelid Anatomy

The regional anatomy and physiology is reviewed in order to better understand the causes and treatment of ptosis. The upper and lower palpebrae (eyelids) are modified folds of skin that can close to protect the anterior eyeball. Six principal planes of tissue form the palpebrae[2]. They are (1) the skin and subcutaneous tissue, (2) muscles of protraction (orbicularis oculi muscle), (3) orbital septum, (4) muscles of retraction, (5) tarsus, and (6) conjunctiva (Figure 1).

The skin of the palpebrae is thin, loose and elastic. Unlike most other regions of the body, it contains few hair follicles and subcutaneous fat. The orbicularis oculi muscle is the main muscle of protraction (closing the lids). It is innervated by the facial nerve (CN VII). The orbital septum is the fascia layer that serves as a barrier between the lid and the orbit. The retracting muscles of the eyelids are innervated by the oculomotor (CN III) and the sympathetic nerve fibres. They function to open the eyelid (see Table 1). The tarsus is primarily composed of dense fibrous tissue layer. It provides the main supporting structure of the palpebrae. The palpebral conjunctiva is a layer of mucous membrane which attaches to the tarsus. The most common causes of ptosis involve the muscle of retraction and its innervation.

Classification and Types

There are several classification systems used to describe ptosis. Ptosis may be categorized by age of onset (congenital or acquired)[3]. The most common type of

Table 1: Retracting Muscles of the Eyelids

Eyelids	Retractors	Innervation
Upper	Levator palpebrae superiois	CN III
Upper	Superior tarsal (Muller's muscle)	Sympathetic
Lower	Capsulopalpebral fascia (extension of the inferior rectus)	CN III
Lower	Inferior tarsal	Sympathetic

acquired ptosis results from a disinsertion or rarefaction of levator aponeurosis. The most common type of congenital ptosis results from a poorly developed levator muscle.

Alternatively, ptosis may be categorized by the etiology (myogenic, aponeurotic, neurogenic, mechanical, traumatic)[4,5].

Myogenic ptosis

Myogenic congenital ptosis results from dysgenesis of the levator muscle. Rather than normal muscle fibres, fibrous and/or adipose tissue is present in the muscle belly. This diminishes the ability of the levator to contract and relax. Therefore, cases of congenital ptosis due to maldevelopment of the levator muscle have decreased function, lid lag, and sometimes lagophthalmos (inability to completely shut the eye). Acquired myogenic ptosis is uncommon (e.g. due to muscular dystrophy).

Aponeurotic ptosis

Congenital aponeurotic ptosis is rare and is due to failure of the aponeurosis to insert in its normal position (anterior surface of the tarsus). Acquired aponeurotic ptosis occurs commonly with aging or after chronic ocular inflammation. It is caused by the disinsertion, dehiscence, or attenuation of the levator aponeurosis from the normal position. It may be exacerbated by intraocular surgery. Eyelids with aponeurotic defects characteristically have a higher upper eyelid crease with near normal levator function. Table 2 compares acquired aponeurotic ptosis with congenital myogenic ptosis.

Neurogenic ptosis

Causes of neurogenic ptosis include oculomotor nerve paralysis, Horner's syndrome, myasthenia gravis, and Marcus Gunn jaw-winking ptosis. Oculomotor nerve paralysis may be partial or complete and may be congenital (very rare) or acquired (usually due to trauma). In a complete oculomotor paralysis, the eye is down and out, the pupil is dilated and fixed, accommodation is absent, and marked ptosis of the upper eyelid is present (see Figure 2a). Horner's syndrome is caused by interruption of the sympathetic chain. It is characterized by mild ptosis, miosis, and anhydrosis (see Figure 2b) (see Table 3).

Table 2: Congential Myogenic Ptosis vs. Acquired Aponeurotic Pstosis

	Congential Myogenic Ptosis	Acquired Aponeurotic Ptosis
Palpebral fissure Height	Mild to severe ptosis	Mild to severe ptosis
Upper Eyelid Crease	Weak or absent crease in normal position	Higher than normal crease
Levator Function	Reduced	Near normal
On Downgaze	Lagophthalmos	Lid drop

Myasthenia gravis is due to antibodies to the acetylcholine receptors. This causes abnormal fatiguability of the striated muscle after repetitive contraction. It is characterized by unilateral (or later bilateral) ptosis that worsens as the day progresses but can be improved by resting. Often it is associated with diplopia. Marcus Gunn jaw-winking ptosis accounts for 5% of congenital

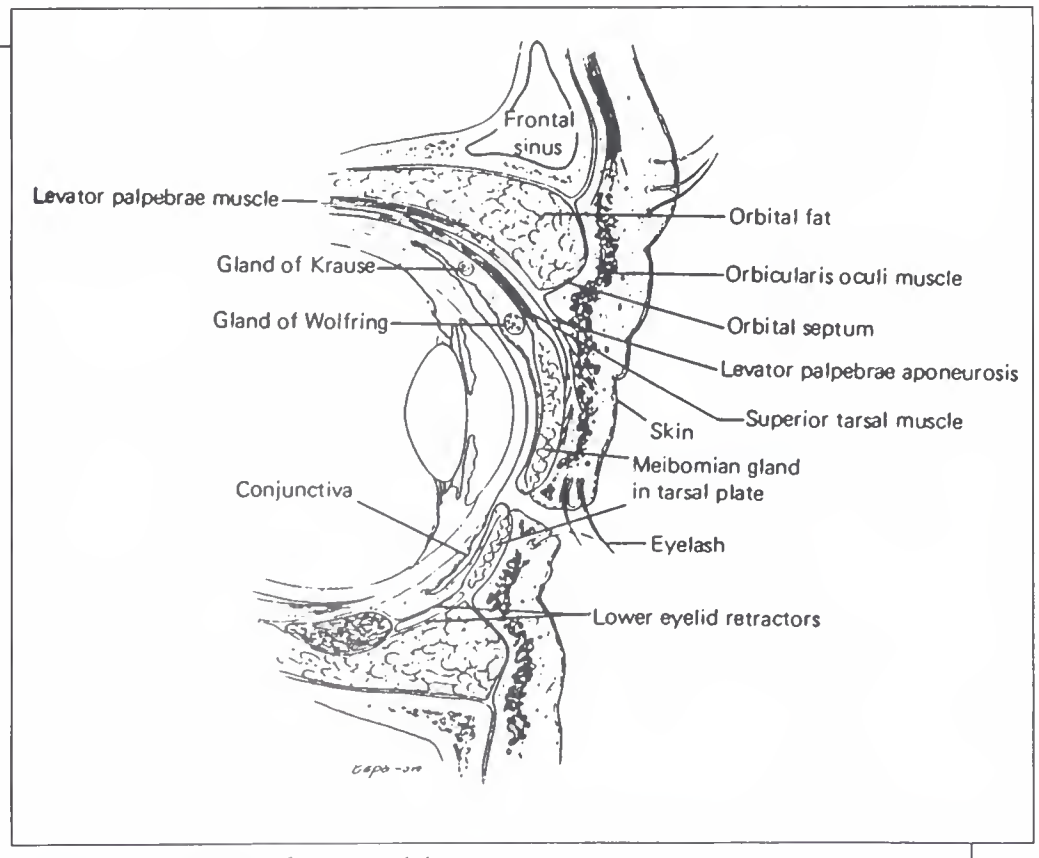

Figure 1: Cross section of the upper lid[1]

ptosis. It is caused by aberrant connections between the motor division of the trigeminal nerve and the levator muscle. It characterized by unilateral ptosis that is relieved by opening the mandible.

Table 3: Horner's Syndrome vs. Third Nerve Palsy

	Horner's Syndrome	Third Nerve Palsy
Degree of Ptosis	Mild	Marked
Pupil Size	Small (Miosis)	Large (Mydriasis)
Associated Findings	Anhydrosis (dry) Rubor	Eye positioned down and out

Mechanical ptosis

Mechanical ptosis may be congenital (eg. plexiform neuroma) or acquired (eg. dermatochalasis). Dermatochalasis refers to an excess of skin in the eyelids commonly seen in the middle aged and elderly.

Traumatic ptosis

Blunt or sharp trauma to the levator aponeurosis or its muscle may result in permanent ptosis. The trauma may be related to orbital or neurosurgical operations. Spontaneous improvement may occur.

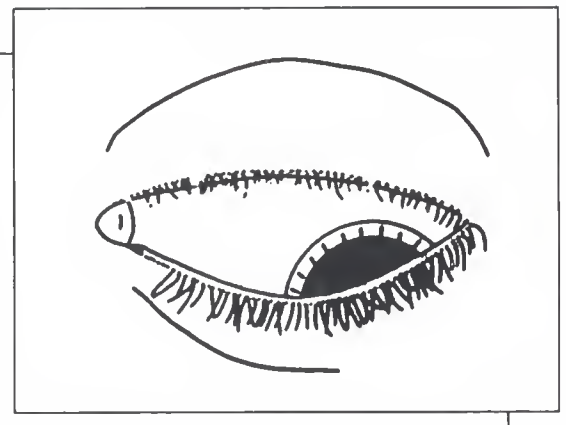

Figure 2a: Complete oculomotor paralysis: Marked ptosis, dilated, fixed pupil; eye turned down and out.

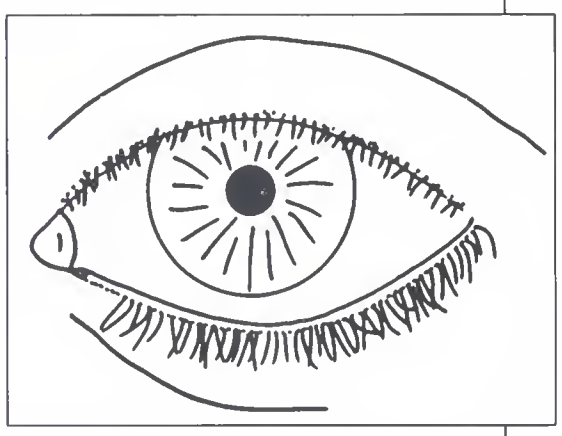

Figure 2b: Horner's syndrome: Mild ptosis, miosis and anhydrosis.

Differential Diagnosis

Several conditions may give the appearance of lid droop when ptosis is in fact not present. This condition is known as pseudoptosis. Causes of pseudoptosis include hypotropia (downward deviation of one eye), enophthalmos (abnormal retrodisplacement of the eyeball), microphthalmos (abnormally small eye), and anophthalmos (absence of a true eyeball)[6].

Evaluation of ptosis

Physical examination of patients presenting with ptosis should include the checking of head positioning, chin elevation, and brow action while attempting upgaze. Four clinical measurements are helpful[7,8]:

1) Palpebral fissure height: The vertical palpebral fissure is measured at its widest point. In children, the palpebral fissure height is normally 9-10 mm. In adulst, the normal range is 10-11 mm[9].

2) Margin-reflex distance: This is the distance from the upper eyelid margin to the corneal light reflex in primary position. It is normally 4-5 mm.

3) Levator function: The levator function is estimated by measuring the upper eyelid excursion from downgaze to upgaze. The normal range for this measurement is 12 -17 mm.

4) Upper eyelid crease: The distance from the upper lid crease to the lid margin is measured. For occidental, the normal range is 8-12 mm. For oriental, the normal range is 3-10 mm[10].

As well, several ancillary tests may be helpful (eg. edrophonium (Tensilon) test for myasthenia gravis).

Treatment and Complications

Various non-surgical and surgical treatment modalities exist for ptosis. Nonsurgical treatment may consist of eyelid crutches that are attached to eyeglass frames. Another simple treatment method involves taping of the upper eyelid open[11]. These techniques provide only temporary relief from ptosis and are impractical.

No effective medical treatment exists for ptosis. One clinically important and common (1:30,000[1]) exception is myasthenia gravis. It can be effectively treated with oral pyridostigmine, an anticholinesterase. Surgical treatment should address the underlying pathological condition. The specific procedure is determined by the amount of levator function present. Most ptosis operations involve resection of the levator aponeurosis or superior tarsal muscle. When levator function is essentially absent, utilization of accessory elevators of the eyelid should be considered. The most frequently utilized accessory elevator is the frontalis muscle.

A basic set of ptosis procedures includes a vertical lid shortening (the Fasanella-Servat operation), a conjunctival -approach levator resection, a skin-approach levator resection (which is best for some cases of severe congenital ptosis), an aponeurosis advancement and tucking, and a brow-suspension ptosis repair[12,13].

Complications of blepharoptosis surgery include undercorrection, overcorrection, unsatisfactory lid contour, lid crease asymmetry, conjunctival prolapse, tarsal eversion, and lagophthalmos with its resultant exposure keratitis.

Summary

Blepharoptosis (ptosis) is abnormally low position of one or both upper eyelids. There are various causes of ptosis, including: Neurogenic (oculomotor palsy, Horner's syndrome, myasthenia gravis), myogenic, aponeurotic, mechanical, and traumatic. Ptosis can be easily recognized and evaluated by general practitioners. It is important to measure palpebral fissure height, margin -reflex distance, levator function, and upper eyelid crease. The patient should be informed about the existing surgical and non-surgical treatment modalities. However, all common causes of ptosis are best treated surgically. One exception is myasthenia gravis, which is effectively treated with anticholinesterase. Effective treatment can minimize the cosmetic and functional impairment resulting from ptosis.

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Pilonidal Sinus Disease: A Review

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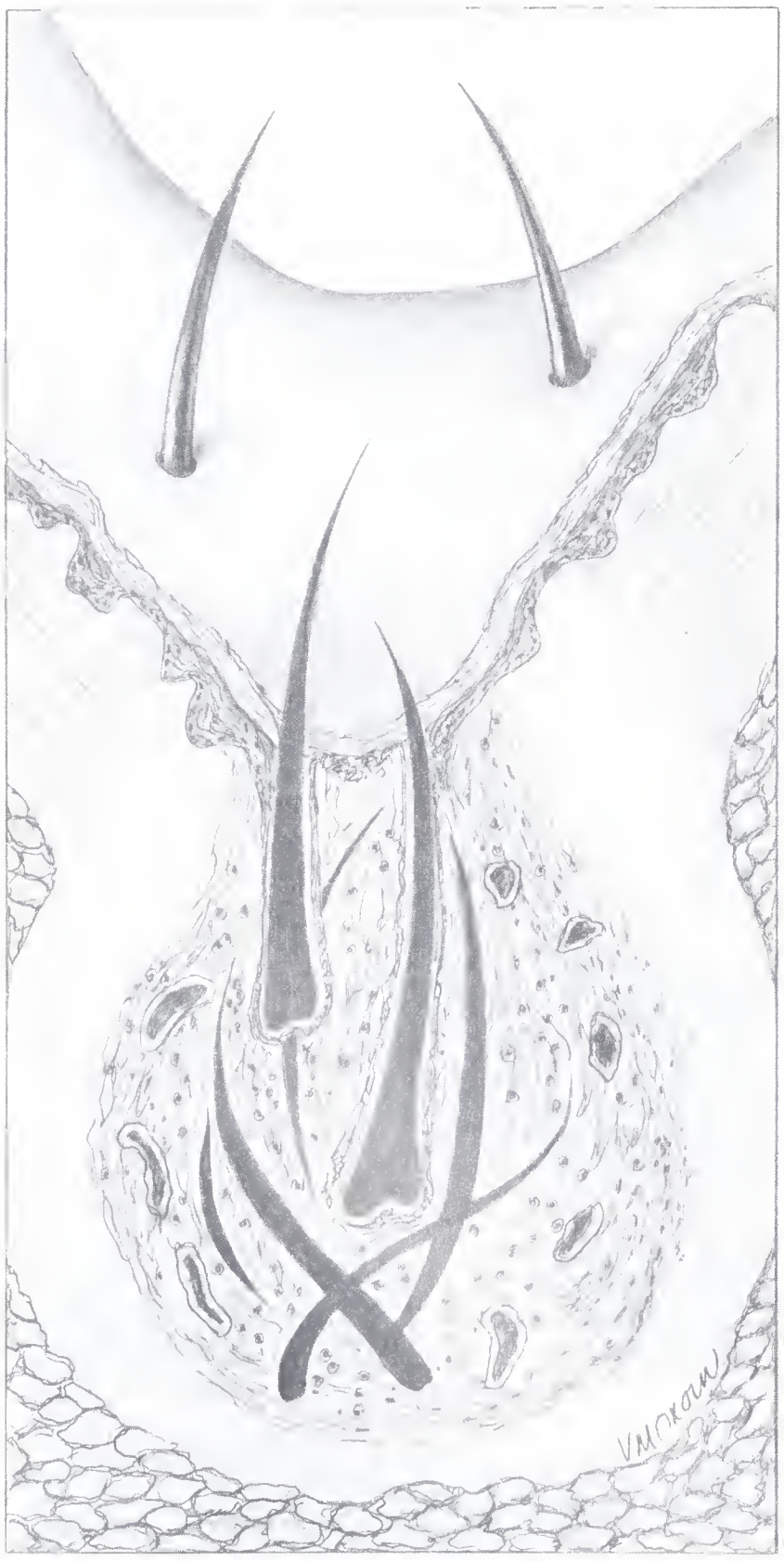

VALERIE OXORN

Abstract

Pilonidal sinus disease of the natal cleft is a very common condition affecting young adults and is a cause of substantial morbidity and lost work time. The pathogenesis of this condition has been a matter of debate since it was first described in 1833. However, it is now widely accepted that the disease is not a congenital condition but originates as a folliculitis which progresses to a foreign body granuloma. The disease also has a high recurrence rate and hence considerable controversy exists about the optimum mode of treatment. In this paper, we review the incidence and presenting signs and symptoms of the disease. We also explore the various theories of pathogenesis of pilonidal disease and the different options available for treatment. Lastly, we discuss complications and the long term prognosis of the disorder.

Introduction

Pilonidal sinus disease was first described by Herbert Mayo 150 years ago[1]. "Pilonidal" is a Latin term derived from "pilus" meaning hair and "nidus" meaning nest. Pilonidal sinuses commonly occur in the sacrococcygeal region[2] but have also been reported in other areas such as the umbilicus[3], chest wall[4], anal canal[5], penis[6], clitoris[7], ear[8], nose[9], scalp[10] and interdigital spaces of fingers[11]. A pilonidal sinus consists of a characteristic midline opening or series of openings in the natal cleft about 5 cm from the anus (Figure 1). Skin grows into the sinus, giving the opening a smooth edge. This primary track leads to a subcutaneous cavity containing granulation tissue and a nest of hairs which may project from the skin opening (Figure 2). Secondary lateral openings are often present, 2-5 cm above the midline pit. Only the skin opening and the superficial track have a squamous cell lining. The deep subcutaneous cavity and the laterally located extensions do not[12].

Pilonidal disease of the natal cleft is very common in young adults but rare in the elderly. In a study of Minnesota college students, its incidence detected on routine physical examination was 1.1% in males and 0.11% in females[13]. The disease is also thought to be associated with sex hormones as it rarely arises de novo after age 40 and has an earlier age of onset in women. Furthermore, the incidence varies among races, being the highest in Caucasians and Mediterraneans and lower in Africans and Asians[14]. This difference is postulated to result from variation in hair characteristics[15] such as kinking, medullation, cuticular scale count, average hair curvature and daily rate of hair growth[16].

The condition is also affected by factors not related to hair characteristics such as obesity[17], poor personal hygiene and buttock friction[18]. Pilonidal sinus disease is frequently referred to as "jeep disease" to highlight the role of occupations like jeep and truck driving which involve the above factors. In fact, Franckowiak has described a hypothetical pilonidal habitus: "... the robust, fat, plethoric type of male with a narrow pelvis, a deep sulcus between prominent folds of the thick buttocks, an excessive glandular activity, and susceptibility to *Staphylococcus* infection[19]."

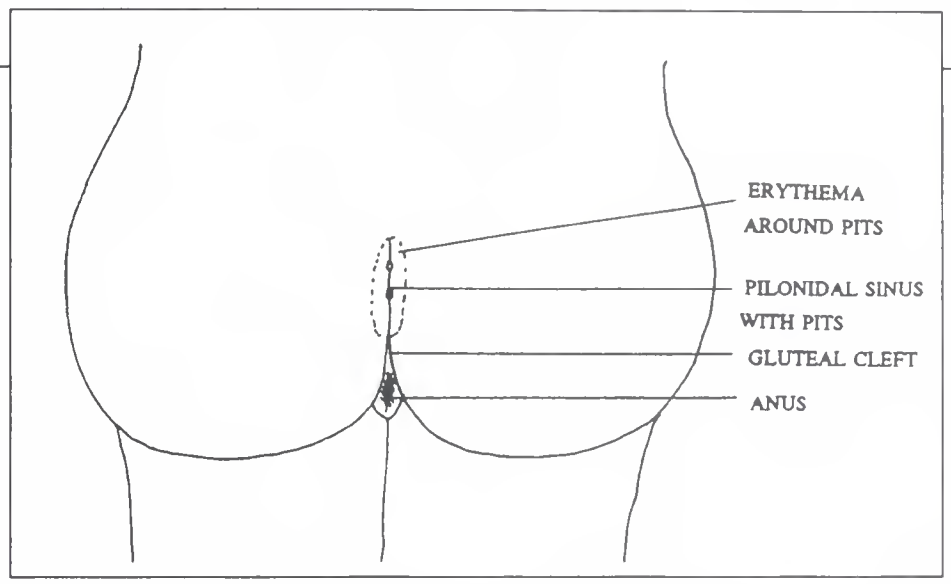

Figure 1: Location of pilonidal sinuses in relation to the anus.

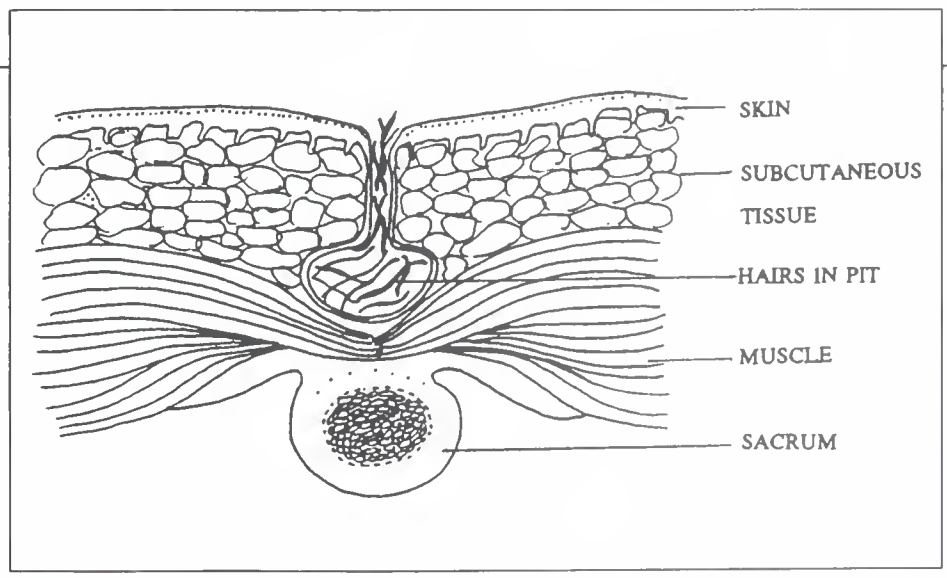

Figure 2: A pilonidal sinus in cross section.

Since the disease was first described, considerable controversy has persisted regarding the pathogenesis and the optimal mode of management of this condition. There are two major conflicting theories of pathogenesis, each with different implications for the extent of resection needed to achieve a cure. We explore these controversies in greater detail elsewhere in the paper.

Etiology

There are two main theories of the pathogenesis of pilonidal sinus disease.

1) Congenital

This theory proposes that the epithelialized tracks found in adult pilonidal sinus disease originate as congenital abnormalities. Proponents of the congenital theory have argued that sacral skin could be indrawn to form a sinus tube by being tethered to the coccyx during growth and that follicles on this skin produced the hair found in the pilonidal sinuses[20]. The congenital theory of pathogenesis was supported by the finding of subcutaneous epithelium-lined spaces (sinuses) in the tissues removed from the sacro-coccygeal region of embryologic and autopsy study specimens[21] and furthermore, by the observation of a familial tendency to pilonidal sinuses[22]. The congenital theory impacts heavily on treatment as it implies that complete removal of all the epithelialized tracks down to the level of the presacral fascia is necessary to achieve a cure. However, this theory has largely been discarded due to a number of reasons. Firstly, congenital neural sinuses are located in the lumbar region and frequently extend to the dura whereas pilonidal sinuses commonly occur in the sacral region and are limited to the subcutaneous space[23]. If congenital neural sinuses occur in the sacral region, they are located more anteriorly than the pilonidal tracks, communicate with the spinal canal and do not contain hair[24]. Secondly, the congenital sinuses are lined by cuboidal epithelium whereas the pilonidal sinus tracts are lined not by epithelium, but by granulation tissue[25]. Lastly, there are no follicles or any other skin appendages in the track lining. This indicates that the hair in the sinus does not arise from the skin of a congenitally derived structure but is of external origin.

2) Acquired

According to this theory, which is currently the most accepted, pilonidal disease starts at the onset of puberty when sex hormones begin acting on the pilosebaceous

Table 1: Factors Promoting Hair Insertion in Pilonidal Sinus Disease

1) **Hair Characteristics**
   a) Number of loose hairs in the cleft
   b) Acuteness of root end of hair
   c) Hair Type (rough or silky, tough more likely to insert)
   d) Hair Shape (straight or curly, straight more likely to insert)
   e) Hair Scalines

2) **Skin Characteristics**
   a) Softness
   b) Macerations
   c) Erosions
   d) Fissures
   e) Pores
   f) Wounds
   g) Scars at cleft

3) Driving Force of Hair into Sinus

   a) Depth of natal cleft
   b) Narrowness of natal cleft
   c) Buttock friction

(Adapted from Karyadakis GE. Easy and Successful Treatment of Pilonidal Sinus After Explanation of its Causative Process. Aust NZ J Surg, 1987; 57:939-42)

units in the natal cleft. With the rapid growth of puberty, stretching of the skin of the natal cleft produces distension of the hair follicles, sebaceous and apocrine glands causing spreading of their orifices on the skin surface[26]. Since anaerobic organisms are normally found in pilosebaceous units, the hair follicle, distended with keratin, becomes infected and the resulting cellulitis produces edema which occludes the follicle opening[27]. The follicle then expands and ruptures into the subcutaneous fat to form an acute pilonidal abscess. Tracks spread out from this abscess cavity in the direction of neighboring hair growth which is cephalad (towards the patient's head) in 93% of cases[28]. The subcutaneous abscess then drains on to the skin surface via a laterally situated tract. As a result, the natal cleft follicle remnant communicates via a track extending from the hair follicle through the abscess cavity to a laterally situated granulating sinus tract opening[29]. A chronic abscess forms when the skin opening of the ruptured follicle opens. Eventually, an epithelialized tube (from the natal cleft opening to the sinus track) is formed by residual epithelial cells at the site of the ruptured follicle.

Therefore, the appearance of enlarged hair follicles is the primary event in the pathogenesis of pilonidal disease. Friction arising from the movement of the buttocks causes suction and exerts a cigarette rolling effect, causing the loose natal cleft hair and other debris to enter the tract each time the patient stands or sits[30]. Three

Table 2 : Treatment Options For Pilonidal Disease

Treatment	Advantages	Disadvantages	Average healing time
1) Closed techniques			
a) Curettage: The midline epithelial follicles are cored out and hairs in the granulation lined track are removed with a small brush. This is repeated at weekly intervals till sinus closes	Outpatient procedure and is performed under local anesthetic	Requires weekly treatment until sinus closes. Regular dressing required	6 weeks
b) Phenol injection: Surrounding skin is shaved and protected with petroleum jelly. Phenol is injected into the track, left in for a minute and then expressed	Outpatient procedure and is preformed under local anesthetic	Needs to be repeated twice	6 weeks
2) Open techniques			
a)Laying open: The pilonidal sinus track is laid open to allow adequate drainage and healing by secondary intention	Outpatient procedure performed under local anesthetic	Granulating wound requires regular out-patient dressing for healing	6 weeks
b) Excision: The midline pits and lateral openings are excised with an area of surrounding skin and the cavity is curetted. Weekly brushing with a small bristle brush is required	Outpatient procedure under local anesthesia	Requires frequent dressing changes	6 weeks
c) Excision and Primary closure: The pilonidal sinus is excised and the wound sutured using deep tension sutures ties over a gauze dressing.	Quicker healing and greater convenience for the patient since no open wound dressing is required	Requires in patient stay and general anesthetic. Delayed healing if wound infected and allowed to heal by secondary intention	2 weeks
d) Excision & Primary Closure with Skin flap: A wide excision of the sinus and tracks is followed by transposition of a skin flap to cover the sacral defect. A myocutaneous flap or a Z-plasty may also be used to close the wound.	Keeps scar away from midline, flattens natal contour, decreases friction	More surgically demanding, requires longer hospital stay and general anesthesia	2 weeks

main factors are postulated to play a role in the hair insertion process. These are summarized in Table 1[31]. Barbs on the hair shafts prevent them from being expelled in the opposite direction and leads to their accumulation within the cleft. The hairs, once trapped, cause a granulomatous foreign body reaction consisting of hair fragments surrounded by foreign body giant cells[32]. Hence, the insertion of hair into the sinuses is a secondary event and tends to prolong disease and delay healing.

The acquired theory of pathogenesis of pilonidal sinus disease suggests that the removal of all presacral tissue in order to remove all embryological remnants of sinuses is not necessary for treatment and cure.

Signs and Symptom

Patients with pilonidal disease may occasionally present with asymptomatic pores or pits in the natal cleft. However, they present with an acute pilonidal abscess in half of the cases. This occurs when an infected hair follicle expands into the subcutaneous tissue or when an acute infection occurs in a pre-existing granuloma[33]. The abscess is classically situated cephalad to the direction of the hair follicle. Pain, tenderness, swelling, heat and a foul smelling discharge are the most common symptoms of pilonidal abscess disease. A small amount of cellulitis and induration may surround the abscess. Fevers, chills, malaise and leukocytosis occur rarely.[14] A pilonidal sinus forms when the pilonidal cavity drains via a sinus track to the skin surface away from the midline in an area of granulation. In some cases, the pilonidal track may run caudally and present as perianal sepsis.[28] In general, symptoms of pilonidal disease fluctuate with recurrences of infection, but are never completely absent between bouts. The diagnosis is made on clinical grounds and there is little need for special investigations. However, pilonidal sinus disease may be confused with a few other conditions which should be considered in the differential diagnosis. These include perianal abscesses (arising from the posterior midline crypt), hidradenitis suppurativa, carbuncles, furuncles and rarely other foci of infection such as osteomyelitis[34].

Pathology

In the earlier stages of pilonidal sinus disease, enlarged hair follicles of different sizes are found in normal looking skin. Each hair follicle contains a single hair shaft surrounded by rings of keratin. When an acute abscess forms, it is usually filled with pus (predominantly polymorphonuclear cells) and surrounded by a wall of edematous fat. When the abscess becomes chronic, it is lined by granulation tissue made up of capillaries, lymphocytes and giant cells. Bacteria like *Staphylococcus aureus* and *Bacteroides spp.* are usually found in the cavity. Chronic abscesses are eventually lined by epithelium that grows in from the skin, forming a cyst.[34]

Treatment

Many different modalities have been advocated for this condition. The various techniques, and their advantages and disadvantages are described below and in Table 2.[11,32,35]

1) Closed Techniques: In these, the skin overlying the sinuses is not incised.

a) Curettage:

This technique, described by Lord and Millar, is carried out as an outpatient procedure[36]. The midline epithelial follicles are cored out under local anesthetic and hairs in the remaining granulation lined track are removed using a small brush . This is repeated at weekly intervals until the sinus closes. This method of treatment can be used only if the sinus is small and not infected. Regular dressing of the site and shaving of the surrounding skin is required. Healing takes an average of 42 days.[32,35]

b) Phenol injection:

After simple curettage, the surrounding skin is shaved and protected with petroleum jelly. Phenol (strength of 50%) is then injected down the track, left in for a minute and then expressed. The process is repeated twice, giving a total exposure of three minutes. Phenol destroys the epithelial component of the track and any debris that may be present within it. A light dressing is applied after treatment. Healing takes about 40 days.[32]

2) Open Techniques: In these, the skin overlying the sinus track is incised.

a) Laying open:

The pilonidal sinus track is laid open under local anesthetic. This allows for adequate drainage and healing by secondary intention. This can be coupled with cauterization of the cavity with diathermy, silver nitrate or freezing. Close supervision is necessary to avoid formation of pockets within the healing wound. Average healing time for this procedure is 43 days but the granulating wound requires regular out-patient dressing for healing.[32,35]

b) Excision:

Under local anesthesia, the midline pits and lateral openings are excised with a small area of surrounding skin and the cavity is curetted. The sinus requires frequent dressing changes and weekly brushing with a small bristle brush. Healing takes about 6 weeks.[35]

c) Excision and primary closure:

The pilonidal sinus is excised and the wound sutured under general anesthesia by using deep tension sutures tied over a gauze dressing. This procedure provides for

quicker healing (2 weeks in 90% of cases) and greater convenience for the patient since open wound dressing is avoided. The disadvantage is that it requires inpatient stay and general anesthetic. Furthermore, if the wound is infected and allowed to heal by secondary intention, healing is delayed. In order to keep the incision away from the natal cleft and to flatten the cleft (to minimize buttock friction), asymmetrical or obliquely placed incisions have been increasingly used as adjuncts to primary closure.[32,35]

d) Excision with primary closure and flattening of the natal cleft:

Skin flaps are used to cover the sacral defect following a wide excision. This keeps the scar away from the midline, flattens the natal contour and hence decreases friction and accumulation of hair. The technique involves excising the sinus asymmetrically with one flap being undermined and translated laterally. A myocutaneous flap or a Z-plasty may also be used to close the wound. This is more surgically demanding and requires a longer stay in hospital and general anesthesia. Furthermore, complications in the form of flap tip necrosis and loss of skin sensation in the flap can occur. Healing time is the same as for primary closure.[35]

The aims of ideal treatment include reliable healing with low risk of recurrence, avoidance of hospital admission and general anesthetic, and minimal patient inconvenience and time off work. None of the described methods completely satisfies these criteria. Closed techniques have healing times and results similar to laying open. Both techniques require long periods of shaving and lavage of the natal cleft. However, in closed techniques, the wound is either small or absent and does not need to be packed open. Both methods are ideal for outpatient treatment but recurrence rates are higher after closed techniques. Primary closure and laying open achieve similar proportions of healed patients 2 months after treatment even though most patients are completely healed within 2 weeks with the former. Primary closure also decreases time off work as patients are not comfortable returning to work with an open wound following laying open. The drawback of primary closure is that hospital admission and general anesthetic are required and the results are surgeon dependent. Primary closure with an asymmetric incision is as successful as simple primary closure but has a lower recurrence rate. Healing by granulation changes the natal cleft anatomy thereby lessening local debris and buttock friction.

Until recently, there were no randomized controlled trials comparing laying open with primary closure techniques. Numerous studies recommended both techniques, but were flawed due to one or more defects in study design like retrospective analysis, not stating patient inclusion and exclusion criteria, short or incomplete patient follow-up and failure to assess patient costs and inconvenience. In the last 4 years a few randomized controlled studies have been completed and indicate that primary closure causes less morbidity, follow-up visits, sick leave and provided early healing when compares to open packing[37,38]. Long term studies will still be needed to see if these results are maintained. We recommend that treatment be individualized for different patients and severity of disease and that laying open be used as primary treatment for patients as this technique has lower rates of recurrence and does not usually involve any hospital stay or general anesthetic necessary for primary closure.

Adjunctive Measures:

a) Antibiotics:

Aerobic and/or anaerobic organisms are found in 50-70% of cases of pilonidal sinus. Clindamycin prophylaxis (effective against both anaerobes and *Staphylococcus aureus*) has been found to reduce average healing time after primary closure. Recently, it has been shown that administration of Metronidazole and Ampicillin intravenously pre-operatively and during the day of surgery is as effective as a 4 day course with these antibiotics in the primary closure of pilonidal and perianal abscess[40]. Routine administration of antibiotics is not recommended if the sinus or abscess is drained and laid open. If the lesion is chronic, the patient has rheumatic heart valve disease or is immunosuppressed, antibiotic coverage for *Staphylococcus* and *Bacteroides spp.* is indicated.[32]

b) Aftercare and Depilation:

Attention to hygiene and shaving of the surrounding area is necessary to prevent penetration of the healing area by hair. If laid open, adequate drainage of the tract is important or stasis and chronic infection will result. Once healing has occurred, continued depilation does not reduce local recurrence because much of the hair that enters the sinus is not locally derived. Attempts should be made to minimize buttock friction and the accumulation of hair and debris in the cleft.[35]

Complications

Squamous cell carcinomas may arise in a longstanding pilonidal sinus. The frequency of malignant change in pilonidal disease is related to the chronicity of the condition and is reported to be 0.1%. Histologically, these tumors are of a high grade and well differentiated. However, they have a clinically aggressive course with recurrences up to 50% and metastases in 14% of patients[41]. They are also very difficult to treat. Local control, for short periods of time can be obtained via radiation. However, radiation is not useful, either alone or as an adjunct to surgery as a method of cure. Wide surgical excision with dissection of clinically involved lymph node seems to be the only reliable method of cure[42]. Reconstruction of the excised area can usually be

accomplished by split-thickness skin grafts. Very large defects may require local advancement or rotation flaps made up of skin or muscle and skin.[41] Giant condyloma acuminatum[43], basal cell carcinoma[44] and adenocarcinoma[45] have also been reported to develop, albeit rarely, in areas of pilonidal disease.

Prognosis

The recurrence rate of pilonidal disease tends to increase with the length of follow-up with most recurrences occurring within three years. Cumulative recurrence increases after one year irrespective of the treatment. Early recurrence (<1 year) is usually due to the persistence of tracks lined with granulation tissue in an incompletely healed wound. Late recurrence (>1 year) is caused by repeated infection of hair follicles. Primary closure leaving a midline scar is associated with a cumulative late recurrence rate of 18% since a midline scar is more prone to subsequent perforation by hair. If the scar is displaced from the midline the rates are less than 10%. The recurrence rate at 1 year following open treatments is 13%. This lower rate has been attributed to a broader scar which has fewer follicles and which flattens the natal cleft to reduce the buttock friction[34].

Summary

Pilonidal sinus disease is a common condition of young adults. It is now well accepted that it is not a congenital condition but is acquired by a series of events which are initiated by the development of enlarged hair follicles in the natal cleft during puberty. Controversy still exists about the optimal course of management of this disease. Although primary closure techniques result in quicker healing, laying open allows for lower rates of recurrence, avoidance of a general anesthetic and a shorter hospital stay. Treatment should be individualized for each patient and measures taken to prevent recurrences.

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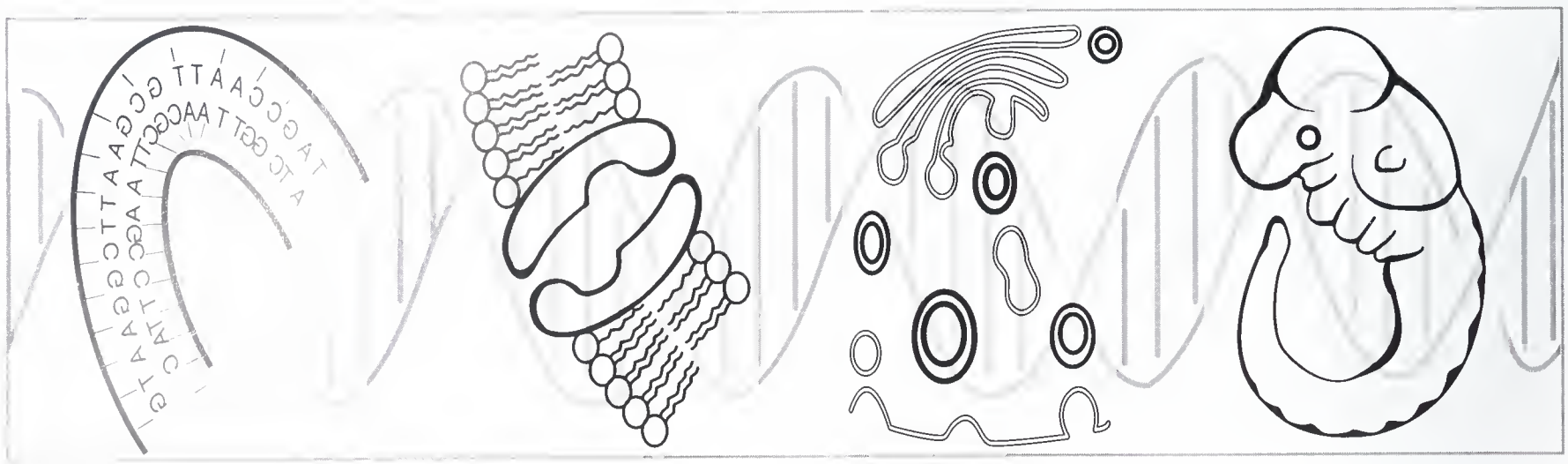
BASIC RESEARCH



JOHN HARVEY

We are pleased to publish the following four abstracts which were selected as the best presentations in the Hospital for Sick Children Research Institute Research Day in August, 1994.

This day was a forum for Summer Research Students to present their work to research staff and other health professionals at the Hospital for Sick Children.

DETECTION OF CYTOCHROME P-4501B1 (CYP1B1) mRNA EXPRESSION IN MOUSE EMBRYOS DURING ORGANOGENESIS

R. Khanna and P.A. Harper, Division of Clinical Pharmacology and Toxicology, The Hospital for Sick Children.

Introduction: The cytochrome P-450 superfamily of enzymes has been implicated in the bioactivation of a myriad of compounds ranging from the endogenous fatty acids and prostaglandins to exogenous drugs such as Nifedipin, Codein, Cyclosporin and environmental agents, such as 3-methylcholanthrene (3-MC), a carcinogenic polycyclic aromatic hydrocarbon (PAH). CYP1B1, described initially by Sutter and colleagues, is a recent addition to this growing superfamily. CYP1B1 shares a 40% amino acid sequence homology with the ubiquitous CYP1A family, which has been evolutionarily conserved, suggesting that they are crucial enzymes in survival and may play a ley role in development. There is little information regarding the regulation of CYP1B1, however CYP1B1 expression is induced by agents such as MC or cigarette smoke in a manner that is analogous to CYP1A induction by these agents. Since P-450s play an important role in xenobiotic metabolism, this study investigates whether CYP1B1 is constitutively expressed and/or is inducible by PAHs during the critical period of organogenesis in mouse.

Methods: Embryos of gestational age day 9.5 (gestational age of 1 refers to the presence of a vaginal plug) were cultured for 48 hr either untreated, in dimethyl sulfoxide (DMSO; vehicle control), TCDD (10 nM), dibenzanthracene (DBA; 10^{-5} M), or 3-MC (10^{-5} M). Total embryonic RNA was isolated according to a modified method of Chomczynski and Sacchi. Following separation of total RNA (10 µg/lane) through a denaturing agarose/formaldehyde gel, the RNA was transferred to a nylon membrane, and hybridized with the 1.0 kB Sma I fragment of CYP1B1 cDNA (provided by Dr. C. Jefcoate, University of Wisconsin, USA). The fragment was labelled with [α-^{32}P]dCTP with a specific activity of > 10^{8} cpm/µg DNA by random priming method (Pharmacia). Specific CYP1B1 signals were detected and quantified by phosphorimaging and autoradiography. RNA loading in the gel was normalised to the amount of actin mRNA in each sample. Embryos were provided by L.Winn (Faculty of Pharmacy, University of Toronto).

Results: Preliminary results from untreated embryos show that CYP1B1 is constitutively expressed at gestational day 11.5. Using Northern analysis we were able to detect CYP1B1 mRNA ~5.2 kB, characteristic of the message for CYP1B1.

Conclusions: Initial evidence suggests CYP1B1 is constitutively expressed in mouse embryos at gestational day 11.5. It is interesting to note that this period coincides with the period of organogenesis in mouse.

(Supported by the Medical Research Council of Canada)

COMPARISON OF FUNCTIONAL CHARACTERISTICS OF THREE ISOFORMS OF THE Na+/H+ ANTIPORTER

S. Wasan and Dr. S. Grinstein, Cell Biology

Introduction: The Na+/H+ antiporter, present in all mammalian cells, has several important physiological roles. Included in these are the regulation of intracellular pH and of cell volume, as well as transepithelial transport of H+ and Na+.

Recent studies have revealed the existence of several isoforms of the Na+/H+ antiporter: NHE-1, NHE-2, NHE-3, and NHE-4. NHE-1 is found in the basolateral membrane of eukaryotic cells, while NHE-3 is present in the apical membrane of epithelial cells. It has not been clarified which of the isoforms are involved in the functions mentioned above. In our studies, several functional characteristics of the first three isoforms were compared. The individual isoforms were expressed in fibroblasts originally lacking Na+/H+ antiport activity.

Methods: The transport activity was followed as the Na+-induced changes of internal pH (pHi) after and acid load, measured fluorometrically in single cells attached to coverslips and loaded with fluorescent indicator BCECF-AM. The exchange was also examined as the 22Na uptake sensitive to amiloride (i.e. a specific inhibitor).

Results: 1) Each of the isoforms was found to be activated by internal acidification. 2) The absence of ATP inhibited the exchange in all isoforms. The mechanism of inhibition was different: in NHE-1 and 2, mainly the H+ affinity of the transporters decreased, while in NHE-3, there was a dramatic decline in the maximal velocity of the transport as well. 3) The osmotic regulation of the NHE-1 and 2 versus NHE-3 was diametrically opposed: the former two were stimulated, whereas the latter was inhibited by an increase in tonicity.

Conclusions: These results imply that each if the isoforms tested can be involved in pHi regulation. Internal H+ exert an allosteric activating effect on all isoforms. Although ATP is not hydrolysed, it is necessary for the normal function of each isoform. The differences in the osmotic behaviour of the isoforms may indicate their distinct function in the body: while NHE-1 and 2 may serve as devices to restore normal cell volume after osmotic shrinkage, the inhibition of NHE-3 by hypertonicity may modulate the transepithelial Na+ transport in a changing osmotic environment (i.e. kidney, colon). This observation may have physiological and clinical implications such as modulation of H+ reabsorption in the kidney, the osmotic diuresis in diabetes, and the pathomechanism of diarrheal states.

DETECTION OF GERMLINE P53 AND P16 GENE MUTATIONS IN AFFECTED MEMBERS OF LI-FRAUMENI SYNDROME-LIKE FAMILIES

C. Tune, M. Goldstein, M. Pinsk (9T7), E. Sexsmith, F. Li,* and D. Malkin. Division of Haematology/Oncology, Department of Pediatrics, The Hospital for Sick Children and *Dana-Farber Cancer Inst., Boston, Mass.

A major player in the pathobiology of cancer is the p53 tumour suppressor gene. Several related functions have been attributed to p53 based primarily on its ability to act as a checkpoint control factor at the G1/S phase of the cell cycle. Suppression of p53 growth regulatory activities via allelic alterations of the gene leads to uncontrolled cellular proliferation. Somatic mutations of p53 are frequently found in sporadic human neoplasms. Recently, another putative cell cycle inhibitor, p16 or MTS-1 (multi-tumour suppressor), has been isolated. p16 mutations have been implicated in a wide variety of cancers. Germline p53 mutations are associated with the Li-Fraumeni Syndrome (LFS), a familial aggregation of diverse cancers that include sarcomas, brain tumours, breast cancer, acute leukemias and carcinomas of the adrenal cortex. Other features of this syndrome include early age of onset of malignancy and the propensity to multiple tumours. LFS is defined by the presence of an affected member with sarcoma under 45 years of age who has at least two first- degree relatives with cancer before the age of 45. LFS-like families are those who, at the time of initial diagnosis, possess some, but not all, of the typical characteristics of classic LFS. The purpose of this study was to determine whether germline p53 or p16 mutations can be detected in members of LFS-like families.

For mutational analysis of the p53 gene, exons 2, and 4 through 11 were each amplified from genomic DNA isolated from whole blood by the polymerase chain reaction (PCR) as fragments defined by various primers specific for each exon. The amplified DNA was screened by single-strand conformational polymorphism (SSCP) using polyacrylamide gels containing varying concentrations of glycerol and bisacrylamide to detect band shifts indicative of single base pair differences. Samples that demonstrated band shifts were subcloned into pBluescript II plasmids and sequenced. Similar techniques were used to identify alterations in exons 1 and 2 of the p16 gene.

Thirteen samples were examined. A germline p53 gene alteration was detected as a band shift by SSCP analysis in 7/13 samples. A germline variation in exon 2 of the p16 gene sequence was seen in 1/13 samples. We are currently completing the sequence analysis of these positive samples.

These results suggest that alterations of either p53 or p16 may occur in LFS-like families and may account for their cancer predisposition.

- Brugieres, L., et al. *Cancer Research* 53: 452-455, 1993
- Vogelstein, B., et al. *Cell* 70: 523-527, 1992
- Malkin, D. *Biochemica et Biophysica Acta* 1994 (in press)
- Kamb, et al. *Science* 264: 436-440, 1994

CONSTRUCTION OF VECTORS FOR THE EXPRESSION OF GREEN-FLUORESCENT PROTEIN IN THE EUKARYOTIC SECRETORY PATHWAY

R. Liu, Dr. M. Manolson and Dr. S. Grinstein, Cell Biology.

Introduction and Background: Due to mutation in the cystic fibrosis gene product, CFTR, it has been hypothesized the poor permeation of counterions leads to a shift in organellar pH. This luminal pH shift in the organelles of the secretory pathway (eg. ER and Golgi) may be the cause of the altered terminal glycosylation that occurs in mucins secreted by CF patients. The purpose of this project is to use the bioluminescent Green-Fluorescent Protein (obtained from Chalfie et al., 1994), as a pH-sensitive in situ means of measuring this deviation in pH. GFP is ideal since its biological reaction does not require any exogenous supply of substrate; rather, it can be excited by a long-wave UV source. In order to use GFP in this investigation, the initial work has consisted of making a series of constructs that would permit the successful transfection of GFP into bacteria and yeast. Yeast has been chosen as the test system due to the simplicity of the molecular biology and genetics involved. Since the yeast vacuole is well defined in terms of its pH and its protein composition, it provides a way of calibrating the pH with the fluorescent signal. Ultimately, if GFP proves effective in yeast, the goal will be to target GFP to the ER and Golgi of mammalian epithelial cells using their respective retention sequences.

Methods: The first construct was made by inserting the DNA encoding GFP into an inducible yeast expression vector. The second construct consisted of cutting the yeast terminator from the previous plasmid and adding it to the 3' end of GFP, thus making a GFP-terminator cassette for future yeast-transfecting constructs (the purpose of the terminator is to ensure the transcriptional machinery stops after the GFP-encoding sequence). Two other constructs have involved the insertion of the GFP-terminator cassette into the vacuole-targeting pCYI-50 and pPRB-Suc2 vectors. These vectors which contain the vacuolar-targeting sequences of carboxypeptidase Y and protease B respectively, are being used in the hope that GFP will be pulled along with these proteins to the vacuole. The first construct was then transformed into yeast and grown on solid and liquid media. The colonies were examined at various optical densities by using i) a long-wave UV lamp, ii) a fluorescence microscope, and iii) a fluorometer.

Results: Initial studies of GFP-transformed bacteria have shown a distinguishable difference from control bacteria under illumination with 365 nm UV light. Examination under the fluorescence microscope and excitation of the cell suspensions with the fluorometer have given variable results. Since the work with the fluorometer has just begun, attempts are being made to find an optimal way of picking up the fluorescent signal from the background noise.

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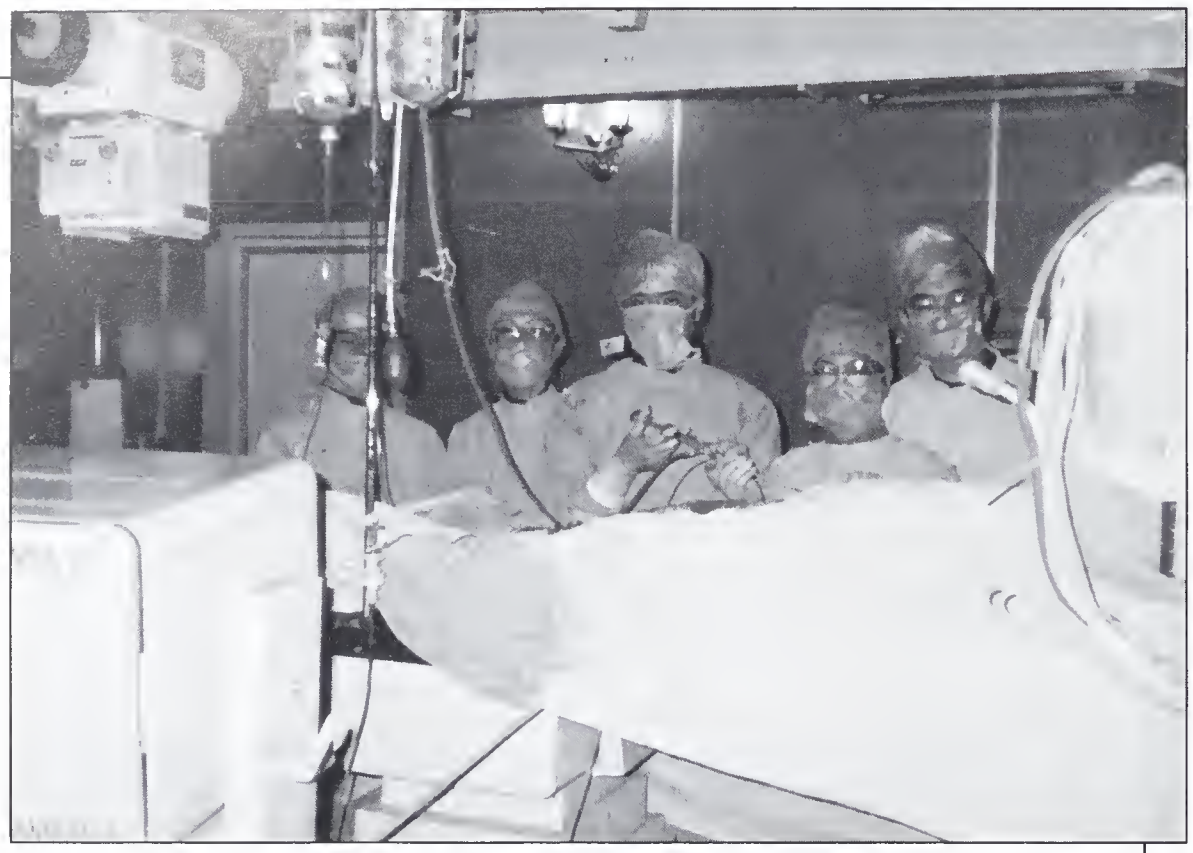
China: Practising First World Medicine in a Third World Environment

Derek So (9T7)

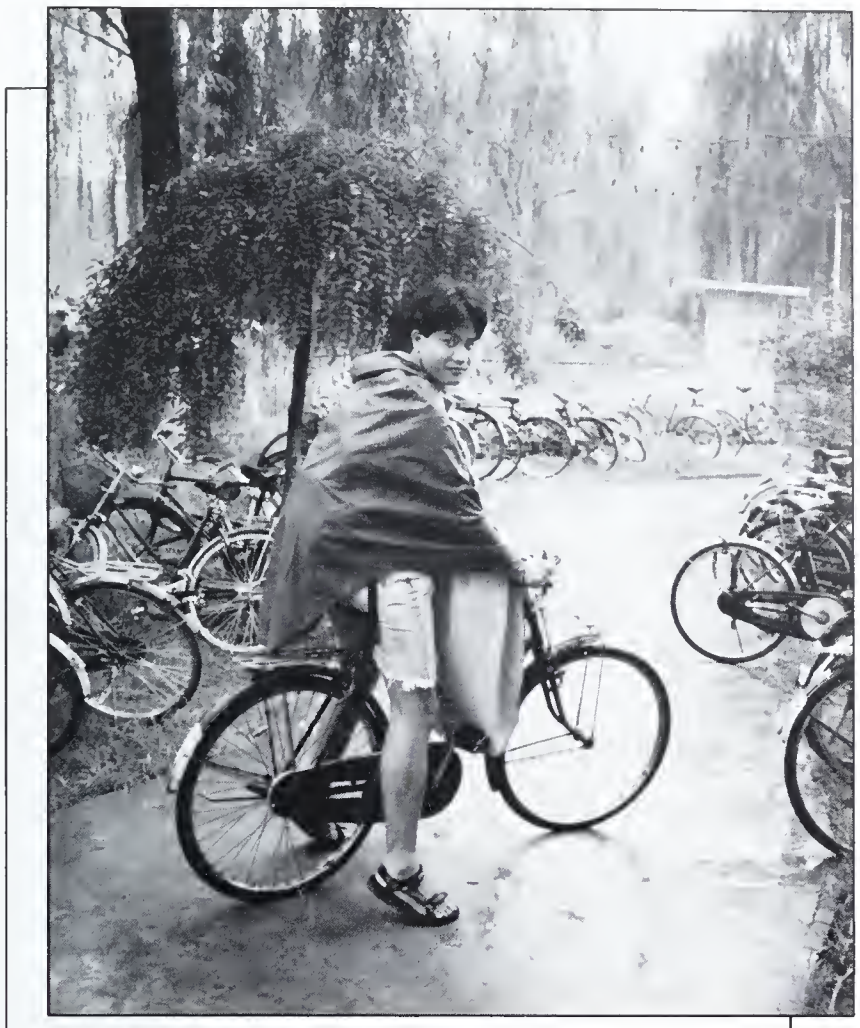


China, a land of rich and diverse history; a land of many mysteries; the land of my ancestors. This was the country I chose to do a project for the International Health Program. Containing close to a fifth of the world's population, China is perceived by many as a developing nation that is quickly approaching the status of a first world country. Western investment in China has helped development proceed quickly. However, the rate of Westernization is progressing at such an alarming rate that one must wonder if Chinese society can keep up. Can medicine be modified to accommodate these changes? Are the Chinese people receiving adequate medical care? These are some of the questions I had as I departed for China. I wanted the opportunity to observe medicine as it is practised outside of Canada and to learn a new language and culture. I knew that my summer in China would fulfil some of these goals.

China is an attractive place to study because it is in a state of industrial and cultural transition. The goal of my project was to study the coronary disease risk factors and how China's progression to a Westernized society correlates with the prevalence of atherosclerosis in the general population . Presently there is an exponential increase in the incidence of coronary artery disease due to atherosclerosis. Thus, China is a good environment to study coronary disease risk factors. This IHP project involved collecting data on patients receiving Percutaneous Transluminal Coronary Angioplasty (PTCA), allowing me to study the incidence of restenosis in these patients.

I worked at the Cardiac Cathetization Laboratory Unit of the First Teaching Hospital of Beijing Medical University. The operating room of the Unit at the hospital was much different from a typical OR in North America. I felt I was in an old movie, sent back to an operating room twenty years ago. The equipment, although in very good condition, was fifteen years out of date. In contrast to North American operating rooms, almost nothing was disposable. Everything from surgical gloves to catheters were sterilized and reused. Watching the nurses prepare the equipment for sterilization made me realize how much waste is generated in North American operating rooms. One must wonder if it's possible to conserve the amount of waste, while maintaining proper measures to combat infection control.

In a developing country such as China, economics dictate the policies and practices in health care. The average worker in Beijing earns about 200 RMB ($33 Cdn) per month. Although the cost of living in Beijing is much lower than in the West, 200 RMB still does not entitle one the standard of living enjoyed by North Americans. For example, many normal citizens have never been out to eat at a restaurant. Contrast this with hospital charges of 20 000 RMB for an angioplasty procedure. Of this amount, 90% of the cost is paid by the employment "unit" of the patient. The remaining ten percent must be paid by the patient. Thus, an angioplasty will cost the patient 2000 RMB, or one year's salary. The hospital takes every opportunity to save the patient money. Catheters, which cost up to 10 000 RMB, are reused, unless a patient's employment "unit"



is willing to pay for the use of a new one. The use of imported versus domestic-made drugs and radiographic contrast medium are also dependent on the patient's financial resources.

One of the most interesting aspects of fugal medicine in China was the use of Ad Hoc PTCA. Ad Hoc PTCA is performed immediately after a coronary angiogram, having never undergone previous coronary angiograms or other interventional treatments. At present, China and Singapore are the only two countries in the world that perform this procedure. The advantages are that it will i) reduce the number of procedures, ii) shorten the hospitalization period, and iii) decrease the cost for the patient. Therefore, this technique not only saves money, but also can be very advantageous for the patient. The main requirements for Ad Hoc PTCA are proper angiographic equipment, well trained support staff and an experienced interventional cardiologist.

One shortcoming of medicine in Beijing is that many of its younger doctors are leaving the country. Doctors in China work long hours, but receive an average wage of 400 RMB ($66 Cdn)/month. Because Beijing is the capital, housing is very expensive. There is also restricted migration into the city. As a result, many doctors are either separated from their spouses or are living in tiny cramped rooms. An opportunity to relocate to a western country, even if it means not being able to be a doctor any longer, is often too tempting for young physicians. Prof. Zhu, my local supervisor, is saddened by this fact. She has dedicated the last ten years of her life to developing future interventional cardiologists for her country. Unfortunately, all her best students have been recruited by American universities.

However, there is still hope for the future of medicine in China. The most inspirational part about my stay was the dedication and spirit of Professor Zhu. She lives with her husband and two children in a hospital residence. Their two rooms combined measure less than three hundred square feet. Dr. Zhu received her interventional cardiology training in Paris, and was offered a position in France. However, she realized that there are many poor people in China and very few doctors. She declined the offer, opting instead to return to China to serve her own people and to train young doctors so that they too can help. In addition to putting in fifty hours a week in the OR, she also commits one weekend per month to helping patients in Chong Chul, a poor rural town four hours drive from Beijing. Her spirit to help the poor, and her commitment to the betterment of her fellow countrymen is admirable. Her only hope is that some of her students would stay and become the next generation of interventional cardiologists in China. That way, China can continue to move forward in this ever changing field of medicine.

While in China I also had the opportunity to visit other regions of the country and I was able to visit many different hospitals. I even visited a hospital in Xian where I was shown many incredible demonstrations of traditional Chinese medicine. Although in the same country, the hospitals can vary greatly in terms of their facilities and quality of the staff.

China is definitely struggling to obtain a new identity as a first world country. Its government has spent a lot of money improving cities and attracting foreign business. Unfortunately, little is being spent on medicine. Doctors are making a valiant effort to provide optimum care. Complicated and state of the art operations are being performed, however, equipment is antiquated. At the same time, China's best young doctors are still lured away by the high standard of living in First world countries. The deficiency of modern equipment and the emigration of young doctors threatens the delivery of health care in the future. The People's Republic of China should delve into these issues and improve its programs. At the same time, Western Medicine can learn a lot from China regarding economy in health care delivery, and preserving first world health care quality. Although China has the potential to perform some new and difficult medical procedures, medicine is not delivered in the most effective way. If China placed more emphasis on the quality of medicine, China might achieve the goal of becoming a first world nation.

Neurology and Neurosurgery Rounds

Danyal Ibrahim, M.Sc. (9T7)

The Journal will be offering a prize of **$50** to the reader who submits the correct diagnoses for all of the cases. Responses should be sent to the following address no later than **January 15, 1995.**

Quick Diagnosis Competition
c/o Danyal Ibrahim - Quick Diagnosis Editor
University of Toronto Medical Journal, Room 2141, Medical Sciences Building, Toronto, Ontario M5S 1A8

CASE 1

Ross Fleming, M.D., F.R.C.S.(C)

Clinical History

A 45-year-old lady presented with 2 years history of unpleasant "pulsatile" numb feeling in her left shoulder and arm, and some pain in the back of her neck. She also complained of numbness and tingling in both feet. She had no headaches.

She could not feel vibration from a 128 Hz tuning fork in either leg below the costal margin, yet joint position sense was normal as were temperature, light touch and pin prick. The knee and ankle reflexes were brisk and the plantars were down. The arm reflexes were subdued, the biceps reflexes being absent. There were no cranial nerve signs.

Figure 1. T1-weighted magnetic resonance image of the cervical spines: *saggital view.*

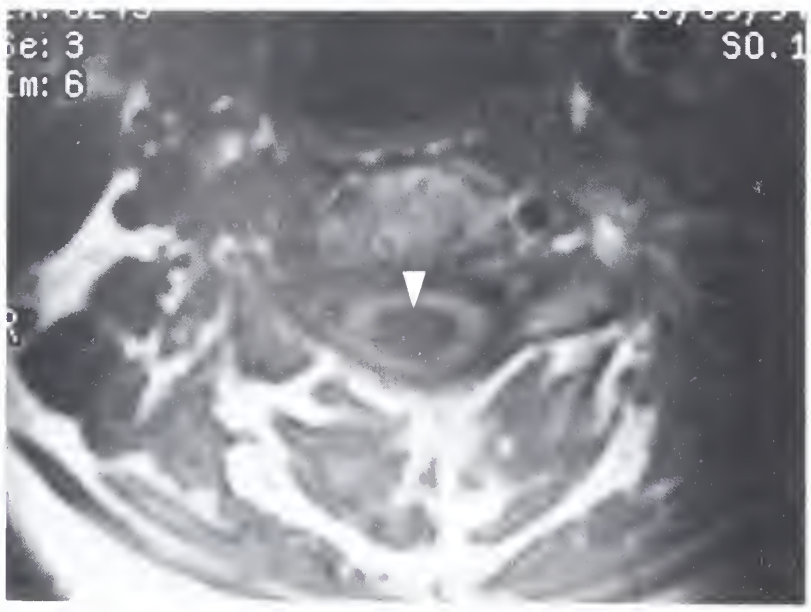


Figure 2. Magnetic resonance image of the cervical spines at C4 level: *axial view.*

What is the lesion indicated by the arrows? What is the most likely cause of this pathology?

Dr. Fleming is a Professor in the Department of Surgery, University of Toronto and a Staff Neurosurgeon at the Division of Neurosurgery, The Toronto Hospital.

CASE 2

John Edmeads, M.D., F.R.C.P.(C), F.A.C.P.

Clinical History

This 49-year-old man presented with a 4 to 6 week history of slowly progressive weakness of his right arm and leg and slurring of his speech. Examination showed mild (grade 4 MRC) weakness in the right arm and leg, spasticity at the right elbow and knee, and bilateral extensor plantar responses.

A CT scan of head and a myelogram were normal. MRI of head is shown. Six weeks after the MRI, as his weakness was improving, the patient died of an acute myocardial infarction.

Figure 1. Magnetic resonance image of the brain: just rostral to the lateral ventricles. *note three discrete focal areas of high intensity (arrow).*

What is the most likely diagnosis?

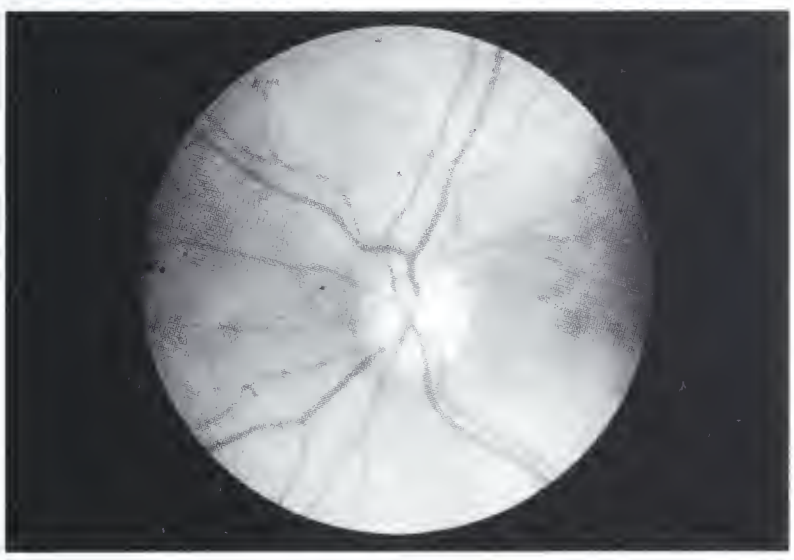


Figure 2. Fundus photograph: *note pale, sharply defined optic disc.*

Figure 3. Autopsy specimen of the brain (formalin fixed): *note periventricular areas of discoloration and sclerosis.*

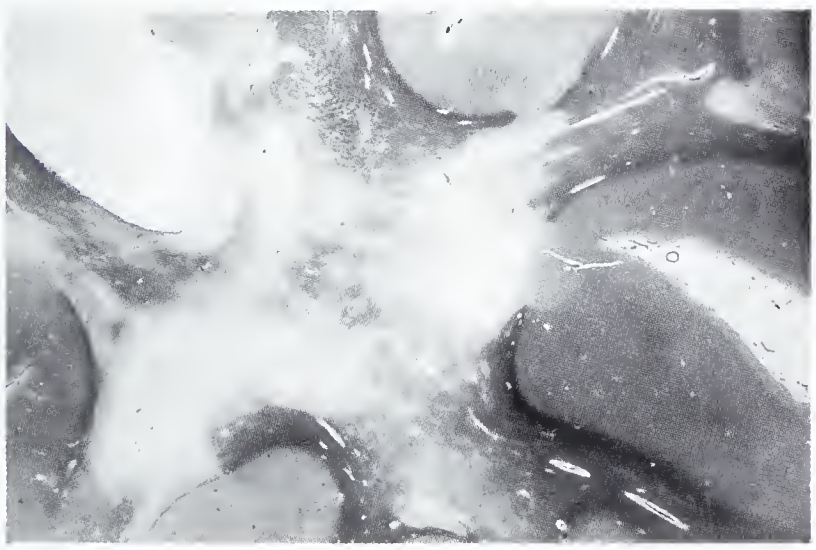


Figure 4. Myelin stain of periventricular region: *normal myelin stains black.*

Dr. Edmeads is a Professor in the Department of Medicine, (Neurology) University of Toronto and Physician in Chief, Sunnybrook Health Science Centre.

CASE 3

Chris Wallace, M.D., M.Sc., F.R.C.S.(C)

Clinical History

This is a 72 year old lady, presenting with 4 months history of slowly progressive visual disturbances in the lateral half of her right eye. She does not have headaches. She has hypertension and is on Atenolol and Novamelore. Her health is otherwise well.

On examination, she is a delightful lady with normal mental status. Cranial nerve examination reveals visual acuity of 20/400 in the right eye and also a very dense right temporal field defect. Pupils are equal and reactive and the right fundus is a little pale. The rest of the neurological exam is intact.

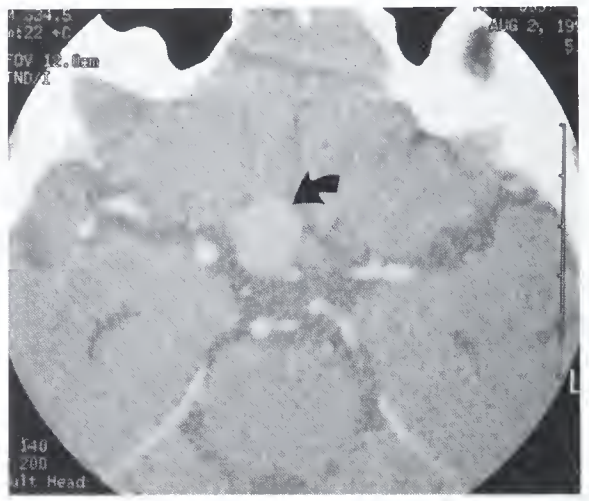
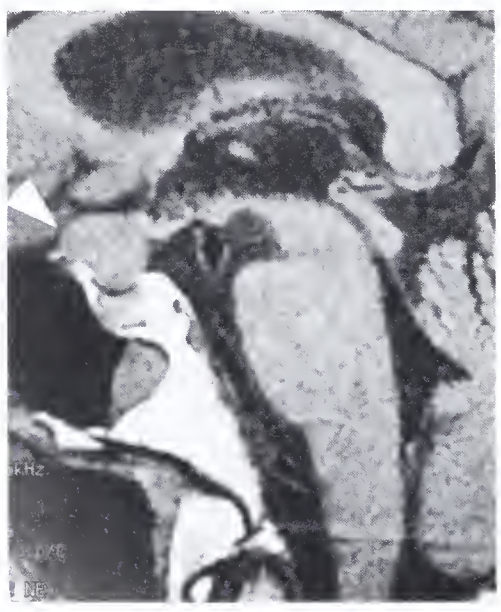


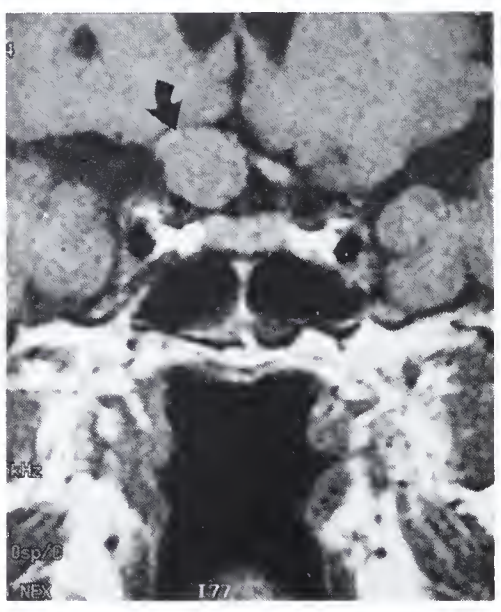
Figure 1. Image from a computed tomography scan of the brain: axial view.

What is the most likely diagnosis?

Figure 2. T1-weighted magnetic resonance image of the brain.



A) Saggital view



B) Coronal view

Dr. Wallace is an Associate Professor in the Department of Surgery, University of Toronto and a Staff Neurosurgeon at the Division of Neurosurgery, The Toronto Hospital.

CASE 4

Ross Fleming, M.D., F.R.C.S.(C)

Clinical History

The patient is 45-year-old man who had new onset grand mal seizure a year and eight months ago.

CT and MRI scans showed a lesion in the right side of the brain. He was started on Dilantin. For the last few months, he had short episodes where he was unable to speak, he had difficulty communicating and difficulty with word finding.

Two months ago, he had a sudden episode of confusion and inability to express himself. En route to the hospital, he had a grand mal seizure lasting 60-90 seconds. He made a fairly quick recovery without neurological deficit and now feels well. His recent MR scan shows a remarkable increase in the size of the right temporal lesion.

Figure 1. T1-weighted magnetic resonance image of the brain: *coronal view (17 months ago)*

What is the most likely diagnosis?

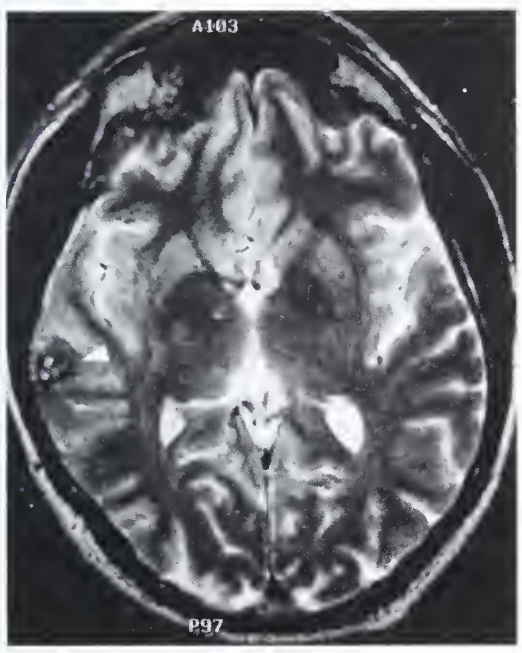


Figure 2. T2-weighted magnetic resonance image of the brain: *axial view (17 months ago)*

Figure 3. Axial gradient echo sequence (MR angiogram): *(14 months ago); no evidence of any abnormal vascularity or flow in the lesion.*

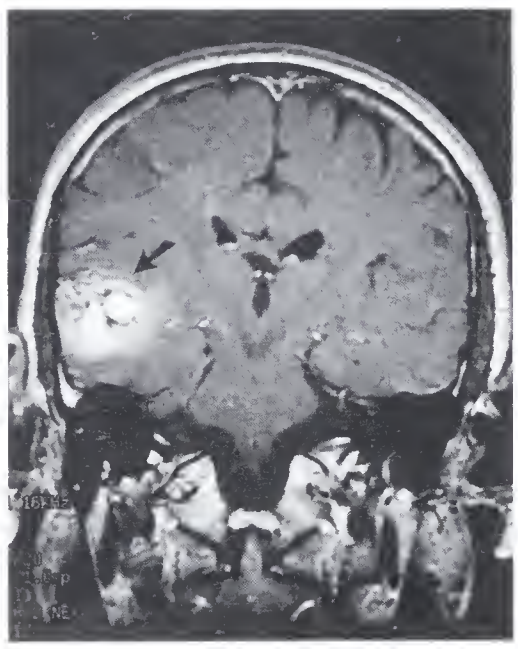


Figure 4. T1-weighted magnetic resonance image of the brain: *coronal view (recent episode); lesion does not show gadolinium enhancement.*

Dr. Fleming is a Professor in the Department of Surgery, University of Toronto and a Staff Neurosurgeon in the Division of Neurosurgery, The Toronto Hospital.

Harrison's Principles of Internal Medicine (13th edition)

Kurt J. Isselbacher, Eugene Braunwald, Jean D. Wilson, Joseph B. Martin, Anthony S. Fauci, and Dennis L. Kasper, editors. McGraw-Hill, Inc. 1994.

Cost: $138 (approx.)

If any textbook makes for bed time reading...it's Harrison's. If its 401 chapters and its 2496 pages won't do it, its sheer weight will subject you into sleep. Like the oracle of Delphi it sits at the centre of our medical curriculum, a sanctuary of knowledge, the ONE you turn to for etiology, pathogenesis, clinical manifestation, treatment etc...If the Gods in the Olympus were to give a modern Hercules 12 labours, one of them would surely consist of reading Harrison's from cover to cover.

Despite its inconvenient size, medical students at any level would feel somehow naked if not armed with the power emanating from its red cover. Although it is probably the most consulted textbook of our academic life, its fragile pages have not been designed to survive the hardships of undergraduate medical education. Maybe that is why every four years there is a new edition.

In addition to being difficult to transport it is also expensive. Approximately $138 plus tax. Just purchasing this book is enough to cause stress ulcers.

Purchasing the newer and improved 13th edition of Harrison's is practically unavoidable. This text is so prevalent in our academic surroundings that it is not surprising that most of us depend on it. Harrison's has been translated into Chinese, French, German, Greek, Italian, Japanese, Portuguese and Spanish; and it is used by medical students all over the world. Its influence on contemporary medical education can be epitomized by the phrase "it is not in Harrison's". Five little words that doom such consequently un-noteworthy information to eternal oblivion.

The book itself is divided into fifteen parts in which the information is classified by organ systems, then by particular diseases. Because the text is written by a number of different authors the format of the presentation varies from chapter to chapter. The first section of every part generally introduces the normal physiology and/or signs and symptoms of dysfunction in the specific organ system. Subsequent sections address systematically specific diseases of the organ system.

It is important to note that the authors clearly did not intend for this text to be utilized for clinical diagnosis. In the clinical learning situation one works from symptoms and signs to diagnosis, while when working from Harrison's it is essential that you know what you are looking for. It is primarily a reference text.

As a reference text it falls somewhere between a basic text and a specialized text. It covers a wide range of information and at times seems frustratingly incomplete, either because you are assumed to have the background or because it presumes what follows may not be pertinent to the unspecialized reader.

It is not the most user-friendly of books; the titles are small, the typeface is small, it can go on for pages without a single diagram, and the only colour brightening the dull demeanor of its pages is a hypnotic baby blue. There are no glossaries. The tables however, are very useful and many of the diagrams are also well organized. There is a list of references at the end of each chapter and despite the fact that this is a new edition many of the references are "classics". This probably reflects the multipurpose function of this text.

Harrison's works best in conjunction with a medical dictionary, a basic textbook (which covers basic principles) and an advanced text or research articles. In this context it is a great text that provides a lot of the necessary information. The essentials of internal medicine are all there, albeit not readily accessible, and it can be used at any stage of your medical education.

The most amazing thing about this book is that it "saves you every time". It shines in your times of direst need. Unfortunately, there is also a "dark side of the force". Having it around makes it so much easier to avoid the library, research articles and more time consuming methods of procuring information. This, however, is highly appreciated in the battle fields of the Medical Science Building.

In addition, the 13th edition caters to the reader of the 90's. Part One, "Introduction to Clinical Medicine" contains new chapters dealing with medical ethics and the impact of social factors on disease. There are new chapters dealing with women's issues and geriatric medicine. Part three "Genetics and Disease" has been extensively reviewed and a new chapter on genes in neoplasia included. Coincidentally, as if in honour of the recent Nobel Laureates, the chapter on G proteins and signal transduction was also revisited.

Major changes were also introduced in the section on "Infectious Diseases" and the star of this section is the new chapter on HIV and AIDS. The section on haematology oncology includes recent advances on oncogenes and tumour suppressor genes and a new chapter on cancer therapy. The section on "Neurological Diseases" boasts a new segment on genetic discoveries in neurology and a

chapter on chronic fatigue syndrome. In addition to the novelties worth highlighting, the core of Harrison's, disorders of the organ system, has also been reviewed.

Despite its pitfalls, the new Harrison's is able to coherently link recent advances in the basic sciences and clinical medicine with those facts that, while not new, remain clinically important. Thus preserving its ranking as the "Citizen Cain" of medical text books.

Claudia C. dos Santos, M. Sc., (9T7)

Pathology (2nd edition)

Emanuel Rubin and John L. Farber, editors.
Philadelphia, J.B. Lippincott, 1994. 1578 pp.
Cost: $90 (approx.)

A quick perusal in the medical section of a university bookstore will reveal a number of different pathology textbooks. Having to choose one typically presents the "If-I-buy-book-A-will-I-regret-not-buying-book-B?" problem that all students face. It is safe to assume that any general pathology textbook will contain the basics (and then some). However, for those who have extensive reading/reviewing to do and are also short on time (i.e. all of us), the way in which information is written and presented can go a long way towards making one book superior to another and thus facilitating the choice of textbook.

Pathology (2nd edition), edited by Emanuel Rubin and John L. Farber, is a visually appealing textbook that is organized in a clear and logical manner. The editors note in the preface that the emphasis is on "graphic pattern recognition". To that end, the book contains hundreds of colour photos and diagrams that serve as an excellent supplement to the text. Rubin and Farber have obviously recognized that graphics are a powerful tool for learning important principles that will remain long after the minutiae have been forgotten.

The thirty chapters of Pathology are organized in a fairly standard way: a progression from classical general pathology (topics such as cell injury, inflammation, repair and regeneration) to systems-oriented pathology. As well, the second edition of this textbook includes a new chapter on cytopathology, recognizing the emergence of this important diagnostic modality.

Each of the 39 contributors to the textbook is a "recognized authority" in his/her particular area of pathology. While it is advantageous to have different chapters written by the relevant sub-specialists, a multi-authored book can sometimes suffer from a lack of fluidity between chapters. Rubin and Farber have dealt with this problem by ensuring that the presentation of disease states in each chapter follows an essentially constant format: definition, pathogenesis, pathology, and clinical features (excellent in allowing the reader to connect pathology to "real life").

Much attention is given in each chapter to gross and microscopic pathological features. In addition, the discussions of pathogenetic mechanisms are thorough and integrate relevant biochemistry, immunology, and molecular genetics. One criticism of the book is that certain topics are not covered in sufficient detail. For instance, no discussion of cell death is complete without dealing with apoptosis; unfortunately, the section on this topic is scanty and other references need to be consulted. It must be recognized, however, that a great strength of the textbook is the avoidance of excessive and tedious detail. Concessions have been made to achieve this, but they do not detract from the book's teaching value, particularly since concepts are reinforced so effectively through photos and diagrams.

The 1994 edition of Pathology contains new information as of 1992 and references are included at the end of each chapter. Most of the review articles listed, however, date from the mid-1980s. Readers who wish more up-to-date information on particular topics, especially in immunology and molecular genetics, are best advised to consult Medline for more recent publications.

Ultimately, the choice of textbook is a personal one which depends in large part on one's learning style, as well as one's learning purposes. Rubin and Farber's second edition of Pathology is an excellent textbook for medical students learning about mechanisms and development of disease. The essentials of pathology are well-delineated and the graphics are relevant and effective; accordingly, this textbook will likely remain a superb reference for the next few years. In short, Pathology is an eminently readable book that is also a pleasure to look at and thus to learn from.

Helen Lee (9T7)

Robbins Pathologic Basis of Disease (5th Edition)

Cotran, Kumar and Robbins, editors.
1994 W.B. Saunders Company.
Cost: $95

Robbins Pathologic Basis of Disease, 5th ed., is a 1400 page textbook of pathology directed towards medical students and residents. With the popularity of the problem-based learning method in the medical curriculum, the value of a good textbook cannot be overemphasized. The ideal textbook would be readable, informative, logical, user-friendly and up-to-date without being encyclopedic.

The Robbins Pathologic Basis of Disease, 5th ed. satisfies most of these criteria, following the success and popularity of its predecessors. The overall presentation of the information is logical and consistent. The first few chapters deal with topics which play a central role in general pathology. The subsequent chapters deal with specific disease processes in a systematic way. At the beginning of each chapter about a particular organ system, there is a brief explanation of relevant normal physiology, anatomy and histology. For each specific disease process affecting a particular organ, the authors discuss etiology, pathogenesis and morphology. The text is supplemented by numerous tables, diagrams and photographs (many in colour). There is an extensive list of references at the end of each chapter with many recent articles in an effort to keep the information both accurate and in concordance with present knowledge.

As an example, Chapter 18 of Robbins Pathologic Basis of Disease, 5th ed. deals with the liver and biliary tract. This chapter begins with a brief description of liver physiology and histology. The authors then list and describe some general morphological features expected with hepatic injury before discussing the effects of liver cirrhosis, bilirubin and hepatic bile formation problems, and hepatic failure. The remainder of the chapter is further divided into less generalized topics such as inflammatory disorders, alcoholic liver disease, and transplantation where more specific liver and biliary disorders are covered. To aid the reader in visualizing the pathological findings associated with many of the described disorders, colour and black and white photographs of gross and histological specimens are appropriately used.

Overall the information is clear, concise and readable. There are points where the condensed, detailed information makes it difficult to read especially for someone unfamiliar to the topic. However reading is made easier by the break up of the monotony of plain text by the appropriate use of photos, tables, diagrams, colour and the occasional humourous comment. Although the authors assume the reader has some prior knowledge of anatomy and histology, they review the relevant background information, for example anatomical features and biochemical or molecular mechanisms, that is required to understand the pathology. Also, in an effort to avoid repetition, and to keep the textbook to a manageable size, many sections will make constant reference to other sections in the book. For example, in the entry on cirrhosis, one has to read two other chapters in the textbook for a more complete story. From a reader's standpoint, it would have been easier to have re-written the material in the same section saving the reader the need to jump around the textbook.

One useful feature of this textbook is that the morphological features for a particular disease are distinctly hi lighted from the rest of the text. This makes using the textbook easier to use as a reference when reviewing pathological features. It would have been nice to see more photographs of specimens and slides as a companion to the morphological descriptions. The table of contents is an excellent summary of the topics covered in the book, while the index appears to be complete. The addition of a glossary of terms would be useful for the reader to help clarify the terminology used in the textbook.

The aim of the authors was to produce a textbook on pathology that would "integrate into the discussion of pathologic processes and disorders the newest established information available - morphologic, molecular, and genetic." and "not to permit the book to become larger and more cumbersome, and yet to provide adequate discussion of the significant lesions, processes, and disorders, allotting space in proportion to their clinical and biological importance." These aims are very difficult to reconcile, but we feel the authors have done an admirable job. In all, the medical student or physician looking for a good pathology textbook as a learning tool and source of reference should consider Robbins Pathologic Basis of Disease, 5th ed. as an option.

Hari Mallidi (9T7) and Barry Chow (9T7)

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PULMONARY TUBERCULOSIS AND THE GENERAL PRACTITIONER

M. S. LAUDER, M.D.

Toronto Hospital for Tuberculosis, Weston

Fifty years ago, when pulmonary tuberculosis was much more widespread than it is now, it was diagnosed and treated at home by the family physician. Tuberculosis sanatoria, and tuberculosis specialists, as we have them to-day, were virtually unknown. There were no X-rays and little bacteriological help, so diagnosis and treatment were empirical.

With the development of sanatoria, the introduction of X-ray diagnosis, and the increasing use of collapse therapy, the treatment of tuberculosis became a specialty, and the general practitioner was gradually relegated to a less and less important role.

As a result of this trend, and of the diminishing incidence of the disease, the general practitioner has little or no opportunity of keeping abreast of the advances in this field, and has little interest in it. This is very unfortunate, as the general practitioner should have a very important part to play in the modern campaign against tuberculosis.

I propose, therefore, briefly to survey the present methods of diagnosis, prevention, and treatment, with special reference to the role of the general practitioner.

DIAGNOSIS

It is essential to have a controlling authority responsible for the overall supervision and co-ordination of the various diagnostic and treatment facilities. This is usually a function of the municipal health department, while the Provincial Department of Health exercises a general co-ordinating authority over the whole province. The diagnostic work falls into two main divisions:

1. *Clinics*

Chest clinics are operated by Sanatoria, general hospitals, and directly by the control authority, and deal principally with three classes of patient.

(a) Ex-patients. Since patients who have been under treatment for tuberculosis frequently relapse, it is very important that they should be re-examined at frequent intervals.

(b) Contacts. People who have been in close contact with known cases of tuberculosis have ten times as high an incidence of tuberculosis as the general population. When a new case is discovered, therefore, every effort is made to examine all the contacts, and they should be followed for five years at least.

(c) Suspects. These are patients who, because of suggestive symptoms, are referred by the family physician.

2. *Surveys*

It is well recognized that a person may have active tuberculosis without any symptoms, and, except for those in this group who may be contacts, they can only be discovered by surveys. Surveys are of two types.

(a) Group surveys. In these a section of the population is investigated which, because of some special factor, is considered likely to show a high incidence of tuberculosis. Contacts are such a group. We know the highest age incidence to be in adolescence and young adulthood, so high schools, universities, and young people's organizations are another such group. Nurses, because of occupation, and Indians, because of race, are other examples. These group surveys may be done by fluorography, or by tuberculin testing, with subsequent X-ray examination of positive reactors.

(b) Mass surveys. The development of the fluorograph permits the rapid X-raying of large numbers of people at very low cost so that the whole population can be covered. This has already been done in Saskatchewan, where about 90% of the population have been X-rayed at an average total cost of about 90 cents per capita. This method is highly effective, but depends on good organization and a high degree of public co-operation, which cannot usually be obtained without an extensive educational campaign. The ideal would be to carry this programme to the point where every one would be X-rayed every three to five years.

Treatment

Treatment of tuberculosis can be summed up in one word—rest. Rest must include physical rest, mental rest, and local rest of the diseased organ.

The principles of physical and mental rest require no elaboration. All I need say is that experience has shown that, with very rare exceptions, they cannot be adequately provided in the home; only in a sanatorium can tuberculosis be properly treated.

The use of surgical methods to provide rest for the lung has steadily increased and technique has steadily improved.

The commonest method has been intrapleural artificial pneumothorax. However, within the past few years, there has been a real change in our attitude towards this treatment. It is no longer considered a safe and simple procedure; but on the contrary is now regarded as a treatment fraught with very real dangers to the patient, and one which requires the greatest skill, judgment and attention. Many new contraindications have now been recognized. The new attitude toward pneumothorax may be briefly summed as follows:

(1) There are types of pulmonary tuberculosis which are not suitable for pneumothorax treatment in that they require permanent, rather than temporary collapse, for example, cavities, and fibro-caseous disease of the apices of some duration.

(2) If pneumothorax is established, it must be done with the proviso that it will immediately be abandoned if it is not satisfactory. Any of the following should be sufficient reason to abandon a pneumothorax.

(a) Inoperable adhesions

(b) Atelectasis

(c) Persistent effusion

(d) Persistent cavitation

(e) Evidence of visceral pleural thickening, etc.

In other words, unless the pneumothorax is perfectly satisfactory, it must be abandoned, for the end results of poor pneumothorax are often worse than those of the tuberculosis.

Extrapleural pneumothorax is now used only where intrapleural pneumothorax is impossible, and thoracoplasty is contraindicated. It is now realized that it is an irreversible procedure, and must be continued indefinitely with positive pressures, or replaced by thoracoplasty.

Phreniclasia has only very limited application. Its popularity has waxed and waned very greatly, but too often it has been done only for the sake of doing something. Its value is difficult to judge since much of its apparent benefit is probably due to bed rest.

Pneumoperitoneum (in conjunction with phreniclasia) is now an obsolete procedure.

Thoracoplasty is the major method of collapsing the tuberculous lung at present. The operative mortality has been greatly lowered now as a result of improved technique—chiefly the doing of the collapse by stages of two or three ribs at two to three weeks' interval. The wide applicability of this operation, not only as a primary elective collapse, but as a means of dealing both with tuberculosis and its complications, and with the problems arising from other forms of collapse, ensure its continued and increasing use.

Lobectomy and pneumonectomy are, for tuberculosis, a new approach and they are still in the process of development, and evaluation. Operative mortality is high but is being greatly lessened. Extirpation is at present done only in cases not suitable for thoracoplasty, such as basal and hilar cavities which do not collapse satisfactorily by any method, and the large caseous nodule, which will eventually excavate but cannot be collapsed until it does. The use of extirpation is limited by the restriction that the remaining lung must be healthy. It must be remembered that extirpation is only one part of the treatment of tuberculosis, and is *not* a curative procedure.

Chemotherapy has as yet no place in the treatment of tuberculosis. None of the agents yet developed has been shown to have any significant influence on the course of human tuberculosis. Undoubtedly, it is only a matter of time until an effective chemotherapeutic agent is discovered; but even then, the killing of the tubercle bacillus will leave a problem in the repair of the damaged lung.

I realize that this has been a very superficial and rapid glance over a large and complex field, but I hope it will be sufficient to give some idea of the achievements in this branch of medicine, and serve as background for my remarks on the role of the general practitioner.

First, it must be realized that this is pre-eminently a field of medicine which demands special training. It requires much practice to be able to interpret a chest X-ray, and a considerable amount of experience to plan a sourse of treatment for a tuberculous patient.

Assuming that the active treatment of tuberculosis is referred to the Sanatorium, there is still an important role for the family physician before and after the period of sanatorium trtaement.

Diagnosis still depends very largely on the family physician, for the majority of the patients are still diagnosed when they develop symptoms. In view of the tremendous importance of early diagnosis, a heavy responsibility rests on him.

I should point out now, that physical examination of the chest, even by an expert, is totally unreliable as a means of diagnosing or excluding tuberculosis. The physician who, after only a negative physical examination of the chest, decides that there is no tuberculosis, is living in a fool's paradise.

If for any reason the doctor suspects tuberculosis, the first step is a tuberculin test. The patch test is only about 80% as accurate as the intradermal so the latter is usually to be preferred. Careful attention to technique is very important. A negative test excludes tuberculosis, with rare exceptions; a positive test merely leaves the possibility. Ninety percent of positive reactors have no demonstrable evidence of tuberculosis, and another nine percent or better have only calcifications, without any active disease whatever. However when the patient has a positive reaction, he must be X-rayed. This can be done free or for, at the most, a nominal fee, at a chest clinic. Here the patient will undergo whatever investigation may be necessary to produce a diagnosis. These facilities are usually available within a reasonable distance, and the careful doctor will make good use of them. If they are not available, have a plate taken, but send it, *with a history*, to a sanatorium or radiologist for interpretation.

Generally speaking, most failures to diagnose tuberculosis are due to (a) failure to think of tuberculosis, (b) too great dependence on physical examination, (c) failure to make use of the available facilities for diagnostic consultation, (d) failure to realize that a couple of negative sputum examinations by no means rule out active tuberculosis.

So much for diagnosis. If the patient is found to require Sanatorium treatment, the family physician must see to it that the patient goes to Sanatorium. This requires a great deal of tact and sympathy, as there is still a great deal of ignorance and fear of tuberculosis and tuberculosis Sanatoria. The doctor can do a great deal to encourage the patient and his family and thereby facilitate the work of the Sanatorium. He can also do much harm by ill-advised pessimism, or by ill-advised optimism. He should not try to ease the shock by saying that the patient will be in the Sanitorium only a few weeks, as this leads to a cruel disillusionment, which greatly upsets the patient. The usual minimum is six months, and the average about a year. The doctor should be perfectly frank, and answer all the questions honestly, without attempting to gloss over the unpleasant details at the expense of misinformation.

Next, the physician should make every effort to co-operate in getting the contacts examined, as this is a very important part of the preventive campaign.

When the patient is discharged from the Sanatorium the family physician is responsible for supervision of his regimen, and for the maintenance of his general health. He should be sure that the patient returns regularly for his review examinations at a chest clinic, as far too many ex-patients drop out of the sight of the Sanatorium once their treatment is complete.

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Reinders FX, Chalin C, and Meier HMR. Alzheimer's disease: Understanding the patient and caregiver. UTMJ 1993; 70(2): 16-20.

For further details, see CMAJ 1985;133:443-448.

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